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STUDIES ON CARDIAC ARREST: THE RELATIONSHIP OF HYPERCAPNIA TO VENTRICULAR FIBRILLATION

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SUDDEN cardiac arrest constitutes one of the chief causes of death during surgery.[7, 8, 11, 16] The precipitating factors of this accident are still obscure except in those instances of overt hypoxia, overdosage of drugs, or peripheral vasomotor collapse. The majority of cases have to be classified as due to undetermined causes. Several observers have suggested that hypercapnia may be one of the responsible factors in this group.[4, 11, 12, 21] A series of experiments designed to study the effects of hypercapnia on the heart have been performed in our laboratory during the past several years.[20, 21] A summary of the experimental findings and their clinical implications form the subject of this paper.

EXPERIMENTAL FINDINGS

Our first efforts to understand the mechanism of cardiac arrest consisted of studies of the effect of vagal stimulation on the heart during hypoxia and hypercapnia.[21] In these experiments 12 dogs were allowed to breathe 20 per cent carbon dioxide and 80 per cent oxygen mixtures. Stimulation of the intact right vagus nerve was carried out until "vagal escape" occurred. The duration of asystole was used as a measure of the cardiac response to vagal stimulation under varying respiratory conditions. In all instances hypercapnia markedly increased the duration of cardiac asystole. In nine subsequent experiments employing respiratory mixtures of 30 to 40 per cent carbon dioxide, it was noted that the duration of asystole varied directly with the degree of hypercapnia. Acute hypoxia, on the other hand, actually decreased the asystole produced by vagal stimulation.

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In the experiments where prolonged hypercapnia with 30 to 40 per cent carbon dioxide was used, the dog's tolerance to severe respiratory acidosis was notable. In these animals, in spite of repeated periods of vagal stimulation, cardiac arrest was never produced. In three instances, however, ventricular fibrillation was noted when after a long period of hypercapnia the dog was allowed to breathe room air. The exact significance of this finding was not appreciated until Brown and Miller[3] made similar observations and reported in detail on this phenomenon.

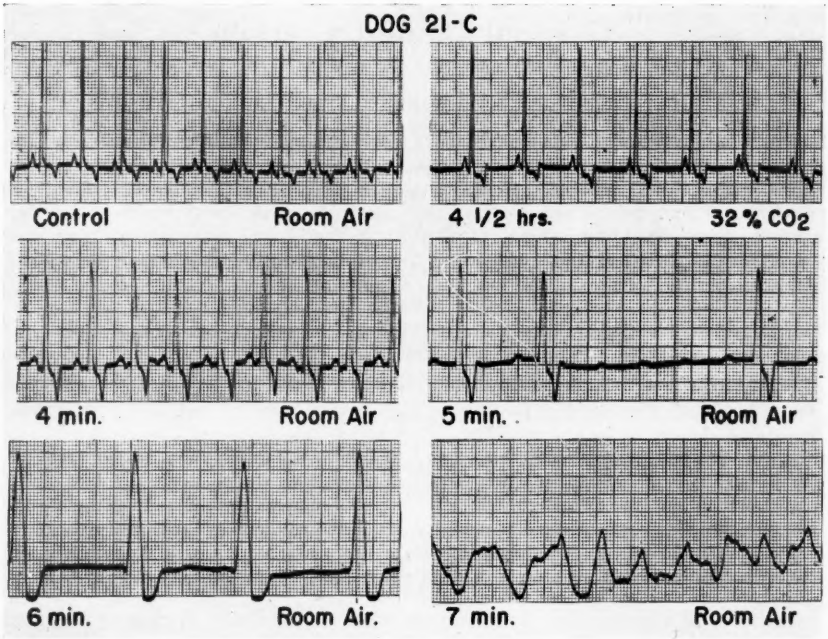

Fig. 1.—Electrocardiogram of a dog showing the development of ventricular fibrillation in the posthypercapneic period.

As shown in Fig. 1, this phenomenon of posthypercapneic ventricular fibrillation is a striking one. Significant alterations in the electrocardiogram always occurred before ventricular fibrillation. These included alterations in T waves, increase in the intraventricular conduction time, ventricular extrasystoles, and A-V dissociation.

Because of the similarity of the electrocardiographic changes of hyperkalemia with those in the posthypercapneic period, another group of experiments were performed, using rats. This afforded an opportunity to study the electrolyte changes in the tissues as well as the plasma. The details of this experiment have been reported elsewhere.[20] These animals were given breathing mixtures containing 35 to 45 per cent carbon dioxide. After periods up

to four and one-half hours, the atmosphere in the chamber was quickly changed to room air. After two minutes the rats were killed, aortic blood was aspirated, and organs to be analyzed were removed.

The results of these experiments are shown on Chart I. The plasma potassium increased in proportion to the length of the hypercapneic period. There was also a significant increase in the level of potassium in the heart muscle, but not in the skeletal muscle, liver, or kidney. In several animals, electrocardiographic changes similar to those observed in the dog occurred in the posthypercapneic period.

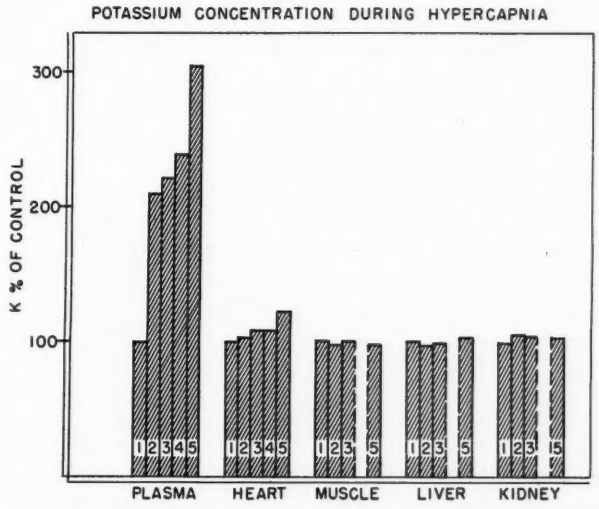

Chart I.—Levels of potassium in the plasma and tissues during hypercapnia. *1*, Data on 14 control rats; *2*, data on 19 animals after 1.5 to 2.4 hours of hypercapnia; *3*, data on 13 rats after 2.5 to 3.4 hours; *4*, data on 5 rats after 3.5 to 4.4 hours; *5*, data on 9 rats that died in chamber after one hour.

Another series of experiments on hypercapneic dogs was then performed, so that serial determinations of the plasma potassium could be followed during hypercapnia and the posthypercapneic period and thus be more exactly correlated with the electrocardiographic changes. The dogs were anesthetized with pentobarbital sodium (30 mg. per kilogram) and given 30 to 40 per cent carbon dioxide in oxygen to breathe. Serial electrocardiogram, arterial pH, and plasma potassium determinations were obtained. After three to five hours the animals were suddenly changed from the carbon dioxide atmosphere to room air and in some instances to 100 per cent oxygen. The plasma potassium concentrations in relation to pH and hematocrit are shown in Chart II. It will be seen that there was an increase in the plasma potassium with carbon dioxide and still a further increase in the immediate posthypercapneic period. Even though some animals were carried for as long as five hours on carbon dioxide, ventricular fibrillation did not occur until the posthypercapneic

period. Chart III shows the plasma potassium changes in 21 dogs. The average rise in plasma potassium on carbon dioxide breathing was from 3.91 to 6.89 meq. per liter with a further increase in the posthypercapneic period to an average of 7.86 meq. per liter. In this group 14 dogs showed significant

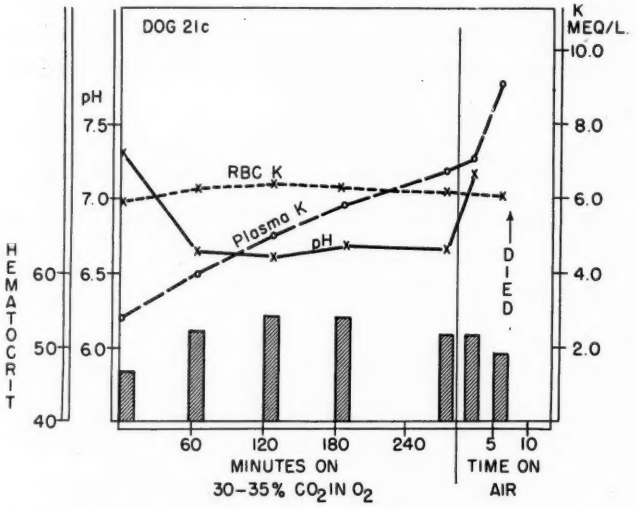

Chart II.—Changes in the pH, plasma potassium, red blood cell potassium, and hematocrit during hypercapnia and the posthypercapneic period.

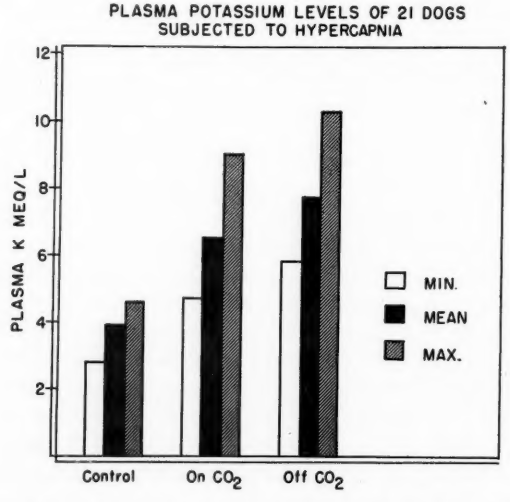

Chart III.—Comparison of the levels of plasma potassium in 21 dogs during hypercapnia and in the post hypercapneic period.

electrocardiographic changes. The average levels during hypercapnia were 7.46 meq. per liter in the dogs showing electrocardiographic changes as compared with 5.73 meq. per liter in the group without electrocardiographic changes. In the posthypercapneic period the group with normal electrocardiograms showed an average rise in potassium to 6.63 meq. per liter, while the other group increased to 8.52 meq. per liter.

A control study of the effect of hypoxia on the potassium level of the blood was done (Chart IV). In this experiment 10 per cent oxygen mixtures were given for thirty minutes, room air for fifteen minutes, and finally 10 per cent oxygen for an hour. There was a slight initial increase in the plasma potassium concentration which soon returned to normal levels.

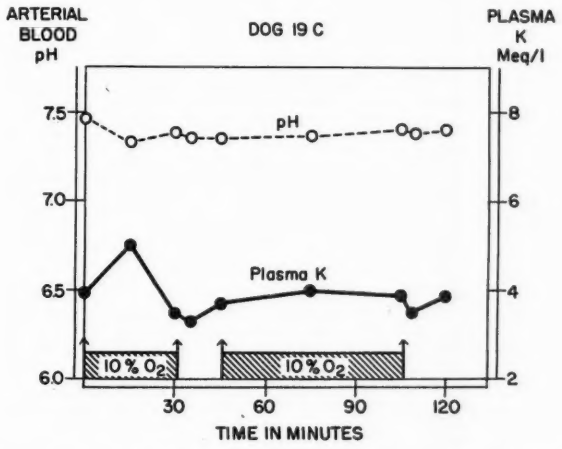

Chart IV.—Arterial blood pH and plasma potassium determinations during intermittent periods of hypoxia.

In another group of 11 experiments blood pressure and respiration tracings were made in order to determine the relation of these factors to the electrocardiogram. In most animals the blood pressure fell just after carbon dioxide breathing was started, concomitant with the initial marked increase in the respiratory rate (Fig. 2). As the amplitude of respiration decreased, the blood pressure rose to levels just above the control. If the animal was carried on to apnea by increasing the degree of hypercapnia, the blood pressure would show a gradual fall. During this period the breathing mixtures contained 60 per cent oxygen administered by positive pressure, so that hypoxia probably did not occur. When the animal was changed to room air, there occurred a great increase in the respiratory rate (Fig. 3). At approximately the same time the blood pressure would fall to one-half to two-thirds the control level, followed in a short time by an increase to normal levels. In two experiments Adrenalin (0.005 mg. per kilogram) given intravenously

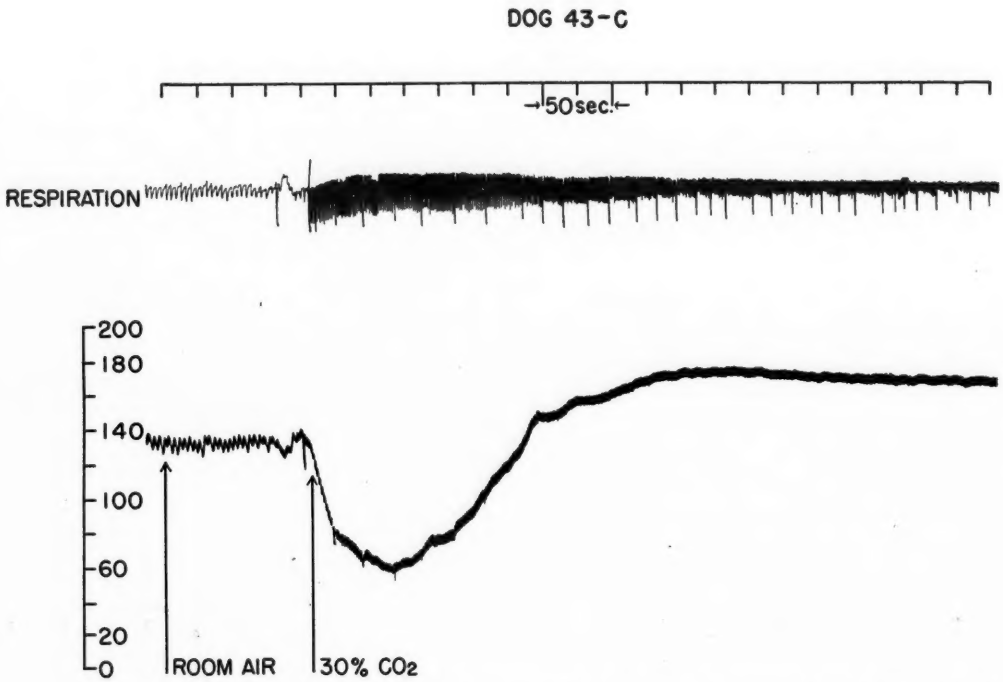

Fig. 2.—A tracing of the change in the blood pressure and respiration on changing from breathing room air to hypercapnia

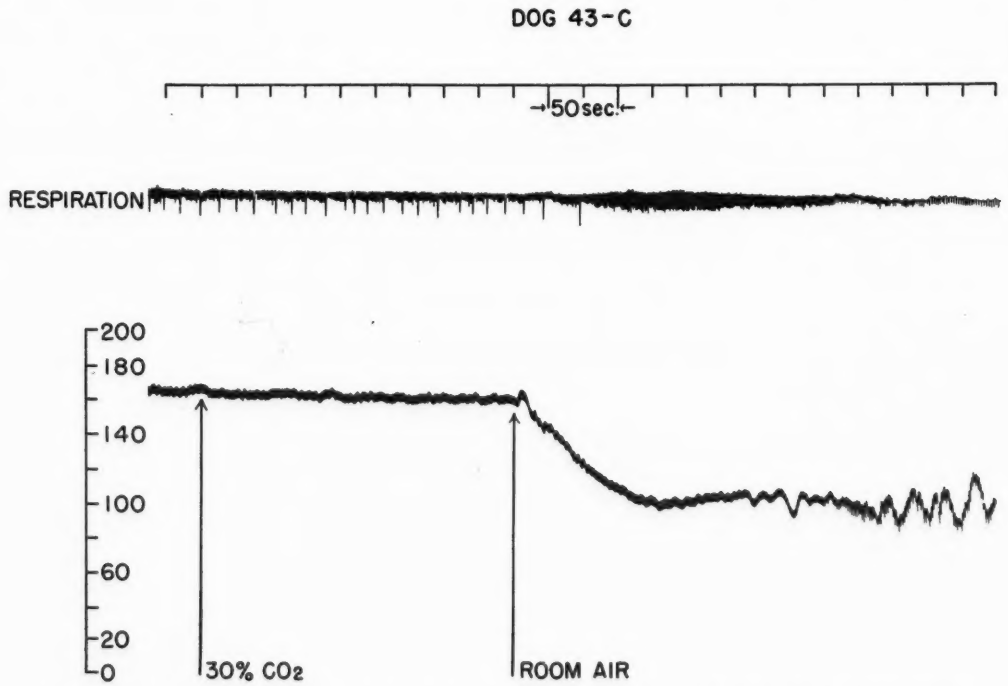

Fig. 3.—A tracing of the changes in blood pressure and respiration on changing from hypercapnia to breathing room air.

immediately prior to return to room air did not prevent the posthypercapneic fall in blood pressure but was followed by a further rise in plasma potassium concentration.

In order to study the relationship between potassium and hypercapnia, a series of nine experiments was performed on dogs continuously infused with 1.14 per cent potassium chloride intravenously at the rate of 1 to 2 c.c. per minute and intermittently made hypercapneic. Serial pH and plasma potassium determinations were obtained. Chart V shows the results of a typical experiment. Breathing of carbon dioxide caused a drop in the pH and a further increase in the already elevated potassium. In a dramatic fashion the

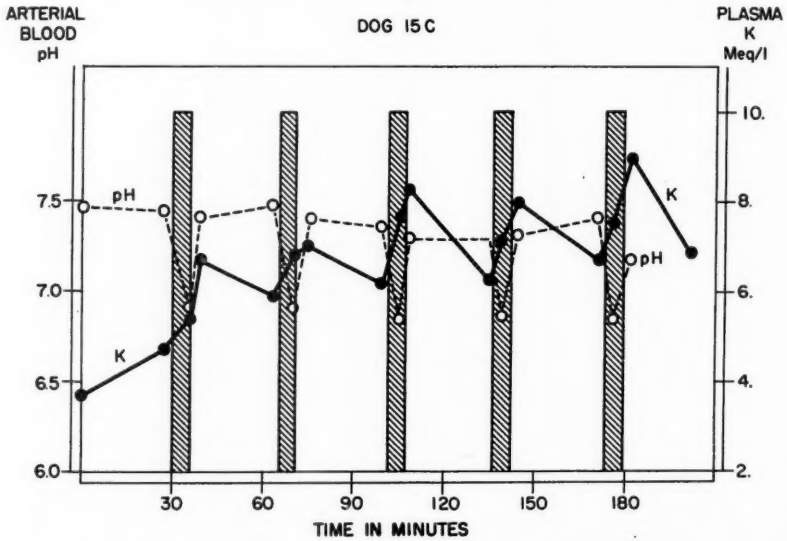

Chart V.—Effect on the arterial blood pH and plasma potassium of intermittent periods of 30 to 35 per cent carbon dioxide breathing in a dog receiving 1.14 per cent potassium chloride at the rate of 1.2 c.c. per minute by the intravenous route.

effect of the posthypercapneic state on the potassium is shown. There was always a further increase in the level of the plasma potassium during this period. In one experiment it was noted that plasma potassium levels of 7.5 meq. per liter were accompanied by more severe electrocardiographic changes in the posthypercapneic period than when the same level of potassium occurred with the animal breathing carbon dioxide or stabilized on room air. The rapidity of the shifts in potassium under hypercapnia were demonstrated, for within three minutes the potassium shifted from 7.5 to 9.0 meq. per liter.

In an effort to reverse the electrocardiographic changes denoting impending fibrillation, hypertonic glucose and saline were given to nine dogs showing these changes in the posthypercapneic period. Both 20 per cent glucose and 3 per cent saline solutions were successful in reverting the electrocardiogram to normal and preventing ventricular fibrillation. Once this latter arrhythmia occurred,

the solutions were without effect. Chart VI shows the effect of 20 per cent glucose on the posthypercapneic rise in the plasma potassium. There was a marked improvement in the electrocardiogram that paralleled the fall in the plasma potassium.

In another attempt to diminish the posthypercapneic rise in potassium, Benodaine,* an adrenolytic agent, was given intravenously to three dogs in doses of 1 mg. per kilogram. When this drug was given just prior to the return to room air, the posthypercapneic rise in potassium was prevented. Benodaine caused a fall in mean arterial pressure to below 60 mm. Hg. No significant electrocardiographic changes accompanied this hypotension.

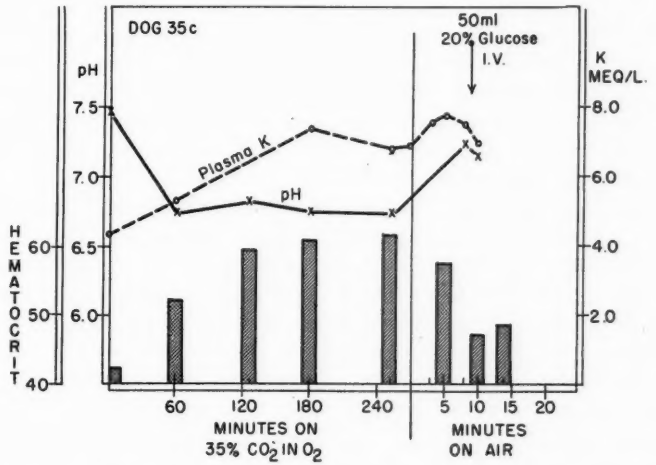

Chart VI.—Changes in plasma potassium, arterial pH, and hematocrit during hypercapnia and in the posthypercapneic period. At the onset of marked electrocardiographic changes, 50 c.c. of 20 per cent glucose was given.

The records of patients in our clinic who developed cardiac arrest were then studied. Of the 36 cases studied, 24 were found to have had cardiac arrest developing from unexplained reasons. The remainder developed arrest from anoxia, drug poisoning, or peripheral vascular collapse. Ten cases of arrest in abdominal operations occurred just at the time the wound was closed or was being closed. This is the time the surgeon usually demands relaxation for ease of closure. This necessitated deepening the anesthesia. The eleven thoracic cases developed standstill during the middle portion of the operative procedure. It was considered possible that hypercapnia could have occurred in twenty-one of these cases. In one case the arrest developed just after the release of an extremity tourniquet that had been in place for forty-five minutes. The other two cases had the onset of arrest during the induction of anesthesia.

*Piperoxan hydrochloride (Merck).

Preliminary studies are now being made to determine if plasma potassium concentration changes of a marked degree occur during surgical operations. Thus far none of the patients studied has developed severe respiratory acidosis. The changes noted have been a drop in the plasma potassium to levels lower at the end of the operation than at the start. It may be that these patients were hyperventilated during operation.

DISCUSSION

The rapidity with which cardiac arrest occurs is the most striking aspect of this clinical phenomenon. This fact has led to speculation that some reflex phenomenon was at fault.[15, 16, 17, 21] This has never been proved. It is remarkable how the sympathetic and parasympathetic nerves can be stimulated with little, if any, persistent effect on the heart. The results of our experiments, as well as those of others, have been remarkable in that cardiac standstill or ventricular fibrillation could not be produced by vagal stimulation alone.[21] Even though hypercapnia accentuated the effect of vagal stimulation on the heart, "vagal escape" always occurred. Acute hypoxia, on the other hand, diminished the effect of vagal stimulation on the heart.

Hypoxia has been implicated as the principal cause of sudden cardiac arrest.[7, 16, 17] Unquestionably hypoxia of severe degree will stop the function of the heart and all other organs as well. In studying our patients' records it was evident that few instances of arrest were due to hypoxia. In our group this was usually the result of long-standing diminished circulation or obstruction to the airway.

The role of hypercapnia in precipitating cardiac arrest has been suggested by several observers.[4, 11, 12, 21] This abnormal respiratory state has been found to occur frequently during operations and is the result of poor ventilation.[2, 16, 17, 18] The toxic effects of hypercapnia on the heart have been poorly understood. In fact, the most outstanding observation in our experiments on hypercapnia has been the tolerance of animals for 30 to 40 per cent carbon dioxide. In man, as well, clinical observations indicate that hypercapnia may be well tolerated. Brown and Miller,[3] in studying this respiratory state, showed that ventricular fibrillation developed in the immediate posthypercapneic period in dogs. This was the first demonstration of a serious deleterious effect of carbon dioxide on the heart.

The marked similarity between the electrocardiograms of hyperpotassemia and those noted in the posthypercapneic period led us to carry out detailed studies of the plasma potassium and tissue potassium changes throughout this time both in dogs and rats.[20] During hypercapnia there occurs an increase in the level of potassium, and there is a further rise in the posthypercapneic period, which may parallel the occurrence of alterations in the electrocardiogram. The most obvious explanation is that these changes are due to the hyperkalemia, though the plasma potassium does not reach levels usually expected to cause ventricular fibrillation.[19] It is possible that the hyperkalemia is merely a part of more profound metabolic disturbance. Some of our studies suggest that

the altered state caused by the hypercapnia may make the heart more sensitive to potassium. Where constant infusion of potassium was given, hypercapnia for a short period of time caused marked arrhythmias of the heart at levels of 9 meq. per liter, which was within the range of potassium levels in the post-hypercapneic fibrillators. This plasma potassium concentration did not cause similar severe arrhythmias in dogs given intravenous potassium chloride and breathing ambient air.

The rise in potassium levels after cessation of hypercapnia may be due to a sudden release of Adrenalin.[5, 9, 10] This, of course, immediately leads to speculation as to the relationship of Adrenalin to ventricular fibrillation when combined with cyclopropane or chloroform. Subsequent experiments have suggested that adrenolytic agents may abolish the posthypercapneic rise in the plasma potassium concentration. Substances that cause a reduction in the concentration of plasma potassium also cause an improvement in the electrocardiogram. The experiments where hypertonic solutions were given when electrocardiographic changes indicated impending ventricular fibrillation support this thesis.

Since hyperkalemia seemed to play a role in the development of post-hypercapneic cardiac arrest, other factors which might raise the plasma potassium were sought. Transfusion of stored citrated blood is a possible source of potassium during operation, for as much as 2 Gm. may be given in a liter of stored blood.[6] The more stored blood is agitated, the more likely it is to have a high plasma potassium content. The hurried administration of large quantities of blood to a patient in whom ventilation and renal excretion are impaired would seem to offer a good opportunity for the occurrence of toxic levels of plasma potassium. In one of our cases cardiac arrest developed immediately after the release of a tourniquet on an extremity. This suggests the possibility that a transient hyperkalemia could have occurred from the release of large quantities of potassium from ischemic muscle. Some drugs such as cyclopropane may also increase the level of potassium indirectly by interference with ventilation. The fact that Adrenalin increased the potassium level suggests why it may sometimes throw a failing heart into ventricular fibrillation.

There are other drugs such as ether that significantly decrease the blood levels of potassium.[1, 13, 14] It is a feasible assumption that this agent may actually protect the heart by reducing the levels of the plasma potassium.

In our study the question has been raised whether or not the changes in the potassium level of the plasma could occur rapidly enough to bring on sudden cardiac arrest. In a matter of two to three minutes the potassium level may increase markedly, as shown in our experiments using carbon dioxide and continuous potassium infusion.

It is obvious that the present methods of treatment of cardiac arrest are not satisfactory and there is little promise for further improvement. If our experiments can be interpreted as demonstrating one of the ways cardiac arrest occurs, can this accident be prevented by utilizing these facts? First, of course, is the prevention of hypercapnia by adequate ventilation. It is of

interest to note that our preliminary studies on patients undergoing surgery have shown a drop in the plasma potassium during surgery, perhaps from hyperventilation or the anesthetic agent used. Brown and Miller[3] have suggested that the posthypercapneic fibrillation could be prevented by gradual, rather than a sudden, return to room air.

The next method of prevention is by constant observation of the electrocardiogram, so that signs of impending ventricular fibrillation can be appreciated early enough for prevention. In five of our clinical cases, where direct observation of the heart was possible, bradycardia preceded the onset of cardiac arrest. Ziegler[22] noted a similar change in the electrocardiogram of cyanotic children who developed cardiac arrest while undergoing surgery. In our experimental animals the electrocardiogram changed very strikingly several minutes before final ventricular fibrillation occurred. This electrocardiographic change was the only sign of impending ventricular fibrillation, for there occurred no change in the blood pressure to shock levels or any fall over that expected after a short period of hypercapnia. The warning electrocardiographic signs occurred early enough, so that they could be reversed with hypertonic solutions of saline or glucose.

It is very tempting to implicate hypercapnia as the cause in many cases of sudden cardiac arrest. In our clinical evaluation it was entirely feasible that hypercapnia could have been in part responsible for the accident in nearly two-thirds of the cases. It may be possible that hypercapnia is only one of several ways the heart muscle can be damaged during surgery to the point that it is no longer able to contract forcefully. Further studies on metabolic changes during surgery are needed. It would seem much more likely that the causes of the so-called undetermined type of cardiac arrest lies within this category rather than from simple reflex changes, hypercapnia, hypoxia, or drug toxicity.

CONCLUSION

1. Hypercapnia has been suggested as a factor in the pathogenesis of cardiac arrest.

2. The critical period for the heart is the immediate posthypercapneic period.

3. Studies of electrolyte metabolism in hypercapneic rats and dogs reveal a constant increase in plasma potassium concentration during this respiratory state. The hyperkalemia is most marked in the posthypercapneic period, at which time serious electrocardiographic disturbances occur.

4. It is suggested that changes in plasma potassium, associated perhaps with more complex metabolic disturbances in the heart, are the primary factors in the pathogenesis of posthypercapneic cardiac arrhythmias.

5. The warning signs of posthypercapneic ventricular fibrillation are easily noted on the electrocardiograph before other evidence of impaired circulation occurs.

6. Posthypercapneic ventricular fibrillation can be prevented by the in-

travenous injection of 20 per cent glucose and 3 per cent sodium chloride when the warning electrocardiographic changes develop.

7. The clinical implications of the experimental findings are discussed.

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DISCUSSION

DR. GEORGE H. A. CLOWES, JR., Cleveland, Ohio.—I want to compliment the authors of this paper on their work on the problem of circulatory collapse during surgery. I wish to cite two additional observations.

As the importance of cardiac arrest and the possible relationship of hypercapnia to this event have become apparent over the last few years, we in our clinic were prompted

to study the total effect of induced carbon dioxide accumulation both in the dog and in man. Last year we presented evidence that the electroencephalographic pattern is changed by increasing degrees of hypercapnia in a fashion similar to that caused by ether as the depth of narcosis is increased.* Ultimately all brain wave potential disappears. If a dog be allowed to continue without brain wave potential caused by high levels of carbon dioxide in the blood for more than fifteen minutes, he will probably not recover. Irreversible damage will have been done to his brain. Both in man and animals, partial pressures of carbon dioxide in the blood sufficient to change the electroencephalographic pattern produce little change in the electrocardiogram. This is in marked contrast to the effect of hypoxia which causes electrocardiographic changes at the same time that the electroencephalographic changes occur. Thus, it would appear that carbon dioxide in toxic quantities affects the brain more than the heart.

In regard to the rise in blood potassium which occurs following the introduction of toxic quantities of carbon dioxide, this is a phenomenon of stress, chemical or mechanical, and may be found in numerous tissues. If the kidneys be removed from a dog, there will be a rise in serum potassium if he is subjected to hypercapnia. But despite the absence of the excretory mechanism, we find that the potassium returns to preoperative levels if the hypercapnia is gradually relieved. This probably represents a restoration of potassium to its normal intracellular position as equilibrium is restored. It is our opinion that the tendency to fibrillate following the sudden relief of hypercapnia is due in part to an imbalance between extracellular and intracellular ionic components. The cause of this may be the greater speed of diffusion of the bicarbonate ion.

DR. N. K. JENSEN, Minneapolis, Minn.—Cardiac arrest actually does occur in human beings with hypercapnia. We had the misfortune, while studying a group of serious respiratory cripples undergoing thoracic surgery by following their arterial pH closely, of removing two patients from the operating table with a pH of below 7, one 6.98 and the other 6.82. We had discontinued the surgery and closed rapidly because of the remarkably low pH. Both patients went into cardiac arrest as they began to blow off their retained carbon dioxide. These cases are of particular significance, as their arterial oxygen saturation had been closely monitored during the entire procedure, and neither had shown any anoxia. The respiratory tracings clearly demonstrated, however, that the exchange was not adequate during the procedure to remove the carbon dioxide, although it was adequate with high concentrations of oxygen to keep the arterial blood level at normal oxygen saturation.

DR. JOHN H. GIBBON, Jr., Philadelphia, Pa.—This paper concerns some fundamental and interesting changes which occur when carbon dioxide accumulates in the body. Dr. Allbritten reported last week to the American Surgical Association that during some twenty major thoracic operations there was no increase in the arterial carbon dioxide tension. The expiratory, as well as the inspiratory phase of ventilation was assisted in all of these patients. Of course what we all want to do is to avoid the changes, reported here, which occur with the inhalation of high concentrations of carbon dioxide. I think we need to pay a good deal more attention to ventilation in thoracic operations.

I am a little confused about one aspect of these very beautiful experiments in relation to the work of Dr. Swan. I was waiting for him to speak, but he did not. We have been shown that breathing a high level of carbon dioxide results in an increase in the concentration of potassium in the plasma and, when the subject again breathes room air, the concentration of potassium in the plasma continues to rise for a short time even though the pH immediately begins to rise. Dr. Swan has reported that in hypothermic dogs, following release of caval occlusion, there was an increased concentration of potassium in the plasma. But he also reported being able to defibrillate hearts by injecting potassium salts into the coronary circulation. I hope Dr. Swan will clarify this matter for us.

*Clowes, George H. A., Jr., Kretchmer, Henry E., McBurney, Richard W., and Simeone, Fiorindo A.: The Electro-encephalogram in the Evaluation of the Effects of Anesthetic Agents and Carbon Dioxide Accumulation During Surgery, Ann. Surg. 138: 558-569, 1953.

DR. HENRY SWAN, II, Denver, Colo.—I have been very interested since I heard Dr. Sealy's previous report on this work, because I consider it extremely important. In the clinical situation during anesthesia where carbon dioxide has been accumulating and then for one reason or another is "washed out," as the anesthetists call it, with oxygen, the exact situation which Dr. Sealy has described is set up. It is during this moment, apparently, that the potassium will rise very high, and cardiac arrest may occur.

With regard to Dr. Gibbon's comment, the potassium level decreases in the serum during hyperventilation. Recently, we have learned also that potassium emerges from the myocardium very rapidly during ventricular fibrillation. If one measures A-V differences in potassium between the femoral artery and the coronary sinus, one finds that in the normally beating heart there is essentially potassium balance, but during ventricular fibrillation there is a great outpouring of potassium from the myocardium as evidenced by a very marked rise in the level in the blood in the coronary sinus. In retrospect this seemed to explain why we could defibrillate the cold heart, or for that matter the warm heart, with the aid of potassium chloride and the use of electric shock, which we were not able to do without the use of the potassium. Potassium chloride, therefore, in our experience, was of no value in preventing fibrillation; but once the heart has fibrillated and has been allowed to fibrillate for a period of time, we feel that a lot of potassium has been lost from the myocardium and for this reason potassium chloride is of aid in defibrillating these hearts.

DR. RALPH ADAMS, Brookline, Mass.—I wish to ask a few questions, hoping that some of the experts in this field will speak about the very important clinical aspects of the problem.

Six days ago, I had the unhappy experience of seeing the heart of an 11-year-old boy go into ventricular fibrillation with cessation of effective output, just as a patent ductus was about to be divided. His operative course had been satisfactory until that time. My first question is: What does one do next, when an hour and fifty minutes later he is still massaging the heart and has not been able to get it to go out of ventricular fibrillation or to restore an effective output even momentarily? I am not an expert in this field, never before being faced with ventricular fibrillation during operation. Although I have treated several cases of cardiac arrest, I believe that is a different type of emergency.

Here are the measures I used over the course of 110 minutes in trying to accomplish defibrillation, while constantly maintaining cardiac massage at a rate of 60 to 70 compressions per minute with the patient in steep head-down position. The heart was flicked with the finger; it was allowed to fill with blood to dilatation and then squeezed vigorously; 0.5 c.c. of 1:1,000 Neo-Synephrine mixed with 9.5 c.c. of 1 per cent procaine hydrochloride was injected into the left ventricle; electrical defibrillation was tried about twenty separate times, with variable voltage and amperage; 10 c.c. of procaine hydrochloride solution (1 per cent) was injected into the right ventricle; 100 mg. of Pronestyl hydrochloride was given intravenously. A second injection of 10 c.c. of 1 per cent procaine hydrochloride was made into the right ventricle.

These things were done, one at a time and several minutes apart, but with mounting discouragement and fatigue, and I found it quite a dilemma to know what to do next. The patient apparently was not dead, and yet there was no good reason to believe him any longer alive. How long does one continue? What does one do next?

After one hour and thirty minutes, a visible sinus rhythm appeared in the left auricle and persisted, but was in no sense transmitted to the ventricle. The ventricles remained in fibrillation. At the end of one hour and fifty minutes of massage and ventricular fibrillation, this child, for reasons unknown to me, restored his sinus rhythm in respect to the ventricles. The heart required intermittent manual assistance for another ten minutes until its muscular power became re-established. The ductus was then doubly ligated, the chest wall closed, and the child returned to his room, where he awakened thirty minutes later, recognized and spoke rationally to his mother. At the present hour, six days later, he is rational and mentally competent, giving us a difficult time from

complications typical of acute tubular nephritis, but from which I feel confident he will recover. I know that others here, as well as myself, would appreciate some practical word from those men present who have had experience in this field.

DR. HOLMAN.—Has anyone any comments or suggestions?

Question.—Will someone tell us how long this can go on? Does it go on usually for ten minutes or for an hour? Is this an unusual case, or have others here had similar experiences?

DR. FRANK L. GERBODE, San Francisco, Calif.—Actually I think this probably is a case that should go on record, because I do not know of another case of ventricular fibrillation which has been in a state of fibrillation for an hour and fifty minutes with survival. Practically all the cases recorded in the literature are of relatively short duration and one either restores the sinus rhythm more or less quickly, or death occurs. I think what you did was undoubtedly right, and the moral of the story is that one must persist in his efforts at resuscitation for at least an hour; the fact that you persisted for an hour and fifty minutes means that we must all profit by your experience and try harder for a longer period of time.

Question.—Do you think it necessary to use so many agents?

DR. GERBODE.—It evidently was necessary to use some of these. I would use procaine amide in any difficult case of ventricular fibrillation if I could not promptly restore sinus rhythm with electrical shocks to the heart. It has been shown experimentally that procaine amide is of aid in defibrillating the heart. The dosage is somewhat in question, but I think 100 to 250 mg. is a good dose in this situation. Whether or not it was feasible in this case, or was correct to use Neo-Synephrine with procaine, as Dr. Adams did, I think is open to question, because it has been pretty clearly shown that Adrenalin does increase the coarseness and degree of fibrillation, and perhaps Neo-Synephrine does the same. Anyone faced with this problem should be rather careful about using Adrenalin when ventricular fibrillation is already present. Was Neo-Synephrine used in the early stages of ventricular fibrillation?

DR. ADAMS.—That was the first thing we used.

DR. W. GLENN YOUNG, Jr. (Closing).—We appreciate the discussants' remarks. I do not know how to answer Dr. Adams' question except to add that there is some evidence that if the experimental animal is placed back on a high carbon dioxide mixture the electrocardiogram will revert back to normal. This is brought out by B. H. Scribner in a paper being presented today in Atlantic City which also confirms our findings of a rise in serum potassium during hypercapnia and a further increase on return to room air. If the electrocardiographic abnormalities are recognized prior to the onset of fatal arrhythmias, the administration of hypertonic glucose or saline may be indicated on the basis of our studies.

We do not know whether the hyperpotassemia is the only cause for the cardiac irregularities, or whether the latter depend more directly on complex changes in cardiac muscle metabolism incident to hypercapnia. In our studies on rats there was a marked difference in cardiac muscle as compared to skeletal muscle. Only the cardiac muscle showed an increase in intracellular potassium during hypercapnia. Our future studies will be directed toward clarifying this point.

In answer to Dr. Swan's remark about potassium leaving the heart during fibrillation, I wonder whether this may simply be a result of the arrhythmia, as it is well known that ischemic muscle releases potassium in large quantities, as does the anoxic liver.

SOME PHYSIOLOGIC CONCEPTS OF HYPOTHERMIA AND THEIR APPLICATIONS TO CARDIAC SURGERY

W. G. Bigelow, M.D. (by invitation),* W. T. Mustard, M.D. (by invitation),** and J. G. Evans, M.D.*** (by invitation)
Toronto, Ontario

BECAUSE of the current interest in hypothermia, it has been considered worth while to review some of the physiologic concepts and assess some of its present application to cardiac surgery. The reduction in oxygen requirements of the body produced by general hypothermia not only allows the surgeon greater leeway in dealing with the poor-risk cardiac patients but permits him to interrupt the circulation for a longer period of time than would be possible at normal body temperature. Unfortunately, the application of this principle to intracardiac surgery is limited at the present time by the fact that body temperatures below 24° C. (75° F.) are poorly tolerated.

Ordinary adult and laboratory animals cannot be cooled safely to a body temperature much below 20° C. (68° F.), although infant and immature animals show a greater tolerance to cold. As shown in Fig. 1, the oxygen consumption steadily declines with a fall in body temperature, provided the shivering reaction is carefully controlled and the arterial oxygen saturation maintained. At 20° C. (68° F.) it is approximately 15 per cent of normal. The picture on rewarming is much the same, with a similar gradual increase in oxygen consumption as the body temperature rises.

Fig. 2 demonstrates a gradual fall in blood pressure in the initial stages of cooling and a more rapid decline below a body temperature of 24° C. (75° F.). The heart rate and cardiac output show a more progressive fall during cooling. The cardiovascular state returns to normal on rewarming. The venous pressure rises slightly during cooling. If the positive pressure artificial respiration is vigorous and over a long period of time, there is a greater rise in venous pressure.[2]

The electrocardiographic tracings show typical changes and revert to normal on rewarming. In a continuous electroencephalographic study[3] during cooling experiments in monkeys (Fig. 3), the wave forms diminished and there was often no evidence of cortical activity at 20° C. (68° F.). No wave forms

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suggesting cerebral anoxia were identified, and the normal tracings appeared on rewarming. Behavior tests in monkeys before and after cooling were similar.

In biochemical studies both Fleming and Swan demonstrated that pH of the blood can be varied within rather wide limits, depending on the rate and depth of positive pressure artificial respiration with pure oxygen[4, 5] (Fig. 4). Animals appear to tolerate low body temperatures better if their pH is main-

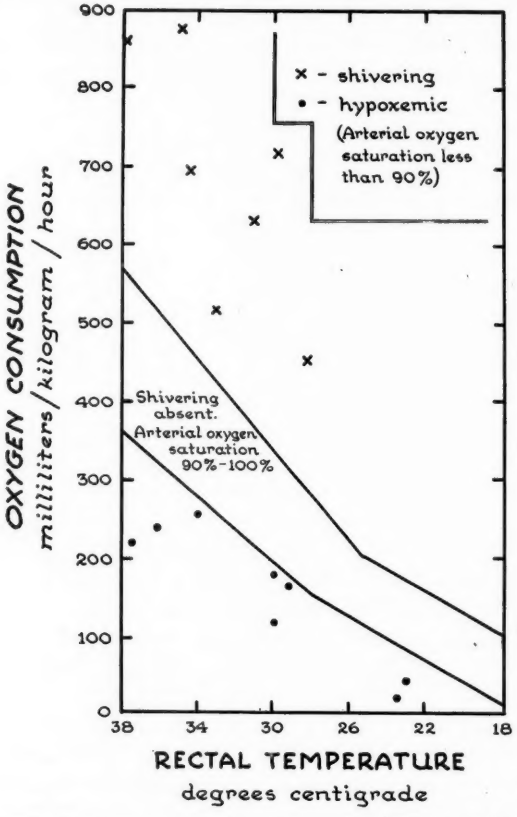


Fig. 1.—A fall of oxygen consumption accompanies reduction of body temperature. All values which were taken under controlled conditions, fell in the lined area. (From Am. J. Physiol.[1])

tained above normal. Swan also noted an interesting drop in serum potassium under the condition of hypothermia plus hyperventilation. He advises the intra-atrial injection of potassium as treatment for ventricular fibrillation.

Since ventricular fibrillation and cardiac standstill are not uncommon complications in hypothermia, and since one or the other invariably occurs if the body temperature is reduced to too low a level, a knowledge of cardiac resuscitation is imperative. To improve our ability to resuscitate animals in cardiac arrest an electrical artificial pacemaker was designed[6] which stimulates

the S-A node and will maintain heart rate and blood pressure in cases of cardiac standstill at low body temperature. This has been combined with a defibrillator in a single resuscitation unit. The electrical pacemaker has not had a satisfactory clinical trial as yet.

Reports from Boerema,[7] Cookson,[8] Delorme,[9] Swan[10] and their co-workers have demonstrated the successful application of the principle of hypothermia

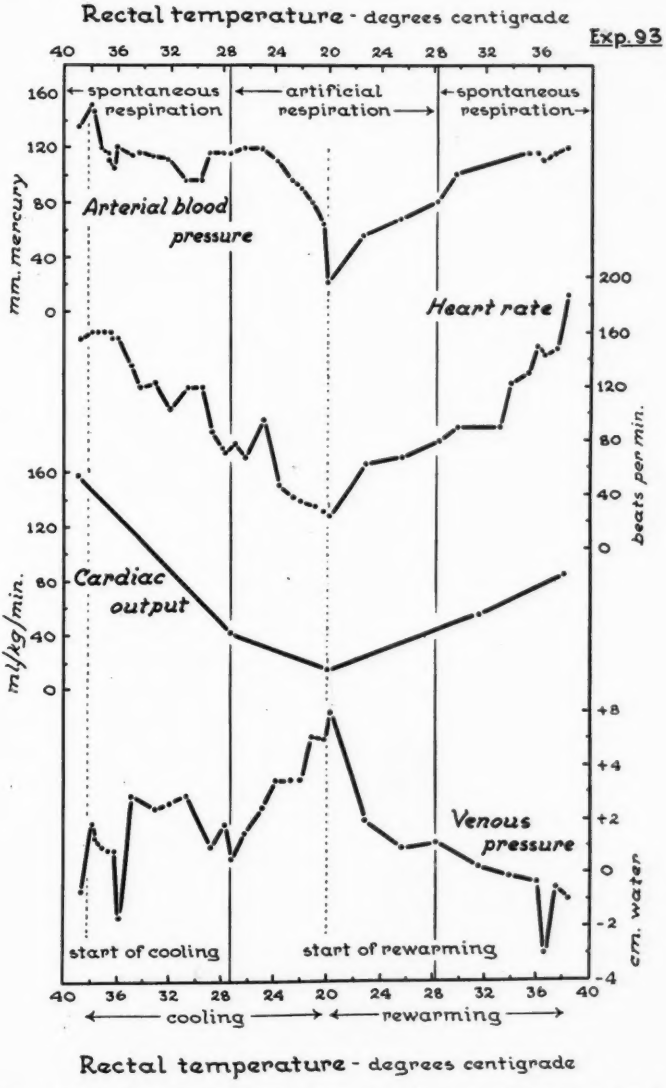

Rectal temperature - degrees centigrade
Exp. 93
40 36 32 28 24 20 24 28 32 36
← spontaneous respiration
← artificial respiration →
← spontaneous respiration →
mm. mercury
160 120 80 40 0
Arterial blood pressure
Heart rate
200 160 120 80 40 0
beats per min.
ml/kg/min.
160 120 80 40 0
Cardiac output
+8 +4 +2 0 -2 -4
cm. water
Venous pressure
start of cooling
start of rewarming
40 36 32 28 24 20 24 28 32 36 40
← cooling →
← rewarming →
Rectal temperature - degrees centigrade

Fig. 2.—Some cardiovascular effects observed during the cooling of a dog to a body temperature of 20° C. (68° F.) with subsequent rewarming. (From Ann. Surg.[2] J. B. Lippincott Co.)

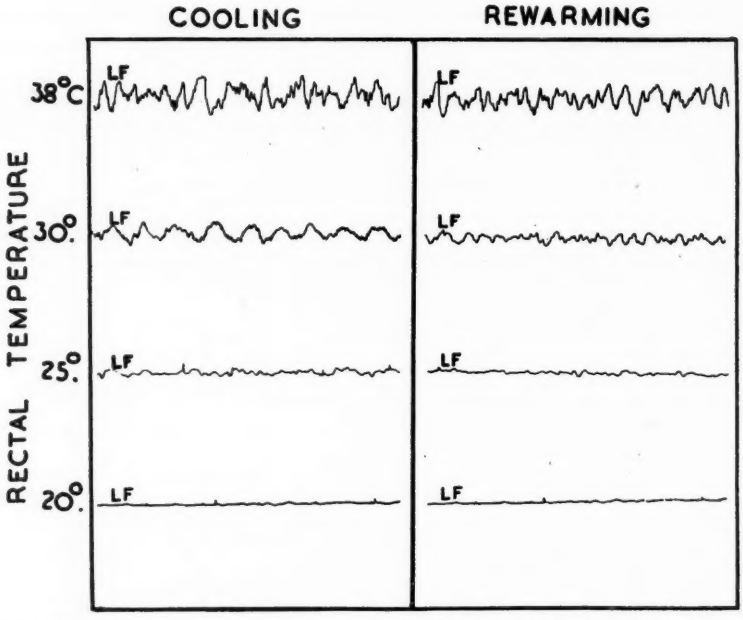


Fig. 3.—Electroencephalographic changes in hypothermia. Changes noted during cooling of a monkey to a body temperature of 20° C. (68° F.) with rewarming. There was a general depression of wave forms with no evidence of cerebral hypoxia and a return to normal on rewarming. (From A. M. A. Arch. Surg.[3])

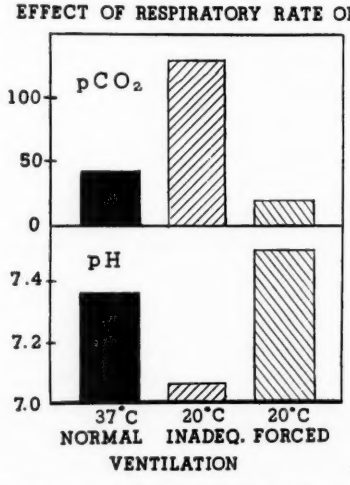


Fig. 4.—A summary of changes that occur in the blood, pH, and CO_2 content at a body temperature of 20° C. (68° F.). There is a wide variation depending on the rate and depth of positive pressure ventilation with oxygen during cooling. (From A. M. A. Arch. Surg.[4])

to experimental intracardiac procedures. It has been demonstrated that a low body temperature increases the time during which the circulation may be interrupted. In monkeys at body temperatures of 16° C. (61° F.) to 19° C. (66° F.), we were able to exclude the heart from the circulation and open it for 15 to 24 minutes, with survival in 12 of 13 experiments.

Hibernation.—The limiting factor in general hypothermia, both experimentally and clinically, is the danger of cardiac arrest at low body temperatures, particularly below 20° C. (68° F.). The influence of age in this matter is interesting since young animals display a much better tolerance to low body temperature; Adolph[11] has shown that infant mammals may be cooled to temperature as low as those that are tolerated by hibernating animals, 3° to 5° C. (37° to 41° F.). We have cooled a 10-week-old puppy to 10° C. (50° F.) with survival, so it would appear that animals, and very likely human beings, lose their tolerance to low body temperatures as they mature. These facts have afforded a stimulus to investigate ways of recapturing this characteristic of the early stages of life. Evidence is available which indicates that human infants possess a similar remarkable tolerance to low body temperature which presumably is lost with age and maturity.

For several years our team has tried various biochemical agents and technical procedures in order to make cooling to low body temperatures a safe procedure and eliminate the complication of cardiac arrest. The influence of such things as balloon obstruction of the aorta, irrigation of the coronary arteries, and exsanguination with retransfusion has been studied. These many tedious experiments have met with only a limited improvement in our technique of cooling.

As another approach to the problem, a study of hibernation has been initiated. The groundhog will not only hibernate at body temperatures as low as 3° C. (37° F.), but he can be artificially cooled to this level by using an anesthetic. At this temperature the circulation has been interrupted and heart opened from one- to two-hour periods on 6 groundhogs with 5 survivals. In an attempt to discover the secret of this unusual tolerance to low body temperatures, biochemical studies were made of the blood both at normal body temperatures and during stages of hibernation.[13] No significant biochemical changes of the order which might explain the above phenomena were found in the blood studies of the groundhog. Thus his unusual cold tolerance has been attributed to a hibernating gland.

Studies of this gland would indicate that it is an endocrine gland of considerable size rather than a storage gland and histologically somewhat resembles the adrenal cortex. There are two large, discrete segments of the gland in each axilla (Fig. 5), but it is diffusely placed throughout the chest and mediastinum, which renders it technically impossible to remove the gland completely from the animal. By cooling groundhogs at various seasons of the year and correlating the results with the size of the hibernating gland, a relationship between the activity of the glands and the animals' ability to tolerate very low temperatures has been found.[14] We have been in the process of ex-

tracting this gland for the past two years, in the hope of obtaining an active principle which, when injected into nonhibernating animals and possibly human beings, will improve their cold tolerance.

Present Research in Hypothermia.—The present-day experimental approach to the problem of direct vision surgery of the heart is being studied with the use of (1) extracorporeal circulation, (2) hypothermia, and (3) a combination of extracorporeal circulation and hypothermia. Those studying hypothermia are lowering the body temperature of animals and human beings by (a) surface cooling and by (b) extracorporeal direct cooling of the blood with or without the use of a pump.

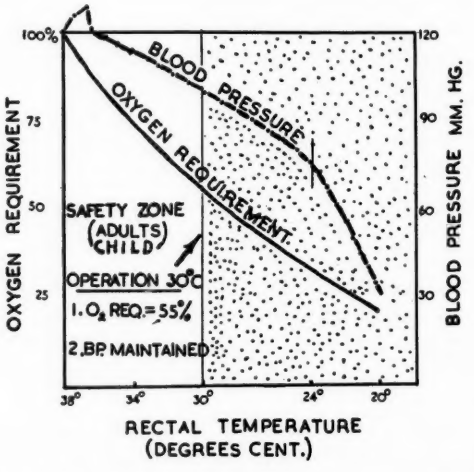


Fig. 5.—Hypothermia for poor-risk cardiac surgery. A graphic illustration of the principles employed in using hypothermia as an adjunct. A body temperature of 30° C. (86° F.) under competent supervision is safe for adults and children. The resulting reduction in oxygen requirements to about 55 per cent with maintained blood pressure appears to reduce the risk of surgery in poor-risk patients. (From S. Clin. North America, August, 1954.)

Extracorporeal circulation studies have reached the stage of successful clinical application by Gibbon and others.

Hypothermia appears to be safe when used as an adjunct to ordinary surgical techniques; but in order to improve the safety of lower body temperatures hypothermia is under study in many centers. No attempt will be made to summarize all the current research that has been reported. To study the mechanism of cardiac failure at low body temperatures, Lange and associates[15] have produced evidence which suggests that this is essentially a hypoxia of the myocardium. Penrod[16] feels that the oxygen utilization of the heart is normal at 20° C. (68° F.). Edwards and associates,[17] working with R. J. Bing, found that the aerobic energy uptake and left ventricular work both decline, but the proportionately greater fall in ventricular work suggested a failure of mechanical efficiency of the heart in the cold state. Hegnauer and Penrod[18] and Juvenelle[19] have reported interesting studies on the cardiovascular dynamics

at low body temperatures. The latter has noted that the myocardial oxygen consumption during ventricular fibrillation in the cold state is not greater than that of normal heart action at the same temperature.

In analyzing the results of studies in hypothermia, it is important to note the body temperatures used in the study. Cookson, Bing, and Swan have made most of their observations at temperatures above 24° C. (75° F.). Our studies would suggest that something unfavorable happens to the animal below 24° C. (75° F.). Therefore it is the range of 20° C. (68° F.) to 24° C. (75° F.) that requires special attention.

In combining hypothermia with extracorporeal circulation, the load upon the oxygenating system is reduced. Juvenelle[19] and Gollan[20] cooled animals to temperatures below the stage of cardiac irritability and arrest, thus allowing a more prolonged interruption of the circulation and subsequent rewarming and survival. Delorme[21] reports that extracorporeal cooling of the blood reduces the "stress" effect of hypothermia. One of us (W. T. M.) is using this combination with a biologic lung oxygenator.

In the study of pure hypothermia, it may be that one will not be able to control ventricular fibrillation and cardiac standstill without a clear understanding of such fundamental problems as oxygen transfer from blood to tissues, and nerve conduction at low body temperatures. Infant animals and hibernating animals may have a similar type of tissue metabolism which may require study.

THE CLINICAL APPLICATION OF HYPOTHERMIA TO CARDIAC SURGERY

There would appear to be two main uses for hypothermia in cardiac surgery: (1) to allow intracardiac exposure with interruption of the circulation, and (2) as an adjunct to render certain surgical procedures upon poor-risk cases more safe. Twenty-one cases of cooling to temperatures of 20° C. (68° F.) to 31° C. (88° F.) are reported.

Hypothermia for Intracardiac Procedures.—Lewis and Varco were the first to report a successful case of intracardiac surgery with hypothermia.[22] Bailey[24] has reported a similar use of hypothermia, and recently Swan[5] has given further impetus to this work by remarkable success in the treatment of interatrial septal defects and pulmonary stenosis by direct vision techniques.

The simplest method of interrupting the circulation through the heart is to clamp the two venae cavae and the azygos vein. Swan[5] points out that this can be tolerated at normal body temperature with no risk for one and one-half minutes. It is usually conceded that a three- to four-minute exclusion of the heart from the circulation is possible with an acceptable risk. Judging from our oxygen consumption studies with a 50 per cent reduction in oxygen-demand at a body temperature of about 28° C. (83° F.), one may expect a relatively safe interruption of the circulation for six to eight minutes. Similarly at 20° C. (68° F.), with the oxygen demand reduced to approximately 15 per cent, a period of twenty minutes should be tolerated by the heart and brain.

In acquired heart disease, intracardiac procedures with hypothermia would not appear to have a significant application at the present time. The common lesions are usually found in adults who tolerate low body temperatures less well, and satisfactory blind intracardiac procedures are being developed. The placing of homologous or plastic valves within the heart under direct vision would require too long a period of interruption of the circulation and too low a body temperature to be tolerated with any degree of safety with our present knowledge.

Congenital heart disease, on the other hand, offers a wide variety of defects and a great deal of scope for the imagination in designing methods of surgical correction under direct vision. In an attempt to analyze the problem of applying intracardiac procedures with hypothermia to congenital heart disease, Table I is presented.

TABLE I. HYPOTHERMIA FOR INTRACARDIAC PROCEDURES

DIAGNOSIS	BODY TEMPERATURE AND PERIOD OF INTERRUPTION OF CIRCULATION LIKELY REQUIRED
1. Complicated Anomalies	
Transposition of great vessels Tricuspid atresia Total anomalous pulmonary venous drainage Etc.	20° C. to 15° C. (68° F. to 59° F.) 15 to 30 minutes
2. Interventricular Septal Defects	
Simple interventricular septal defects Eisenmenger's complex Tetralogy of Fallot	20° C. to 15° C. (68° F. to 59° F.) 15 to 30 minutes
3. Simple Anomalies	
Pulmonary stenosis Interatrial septal defects	28° C. to 24° C. (83° F. to 75° F.) 6 to 10 minutes

The patients who belong in the first group of complicated anomalies are often infants who do not survive much beyond the first year of life. There are several problems which arise in the surgical correction of these defects. They are usually poor surgical risks. Multiple anomalies are common in associaton with the main defect, and accurate diagnosis is difficult. One encounters a very practical problem in attempting to operate upon these patients. The heart is usually so large that it occupies most of the thoracic cage and leaves little room for surgical dissection without preliminary embarrassment of the heart action. Wide exposure, such as afforded by a transsternal incision, is usually required.

The highest incidence of cases comes in the second group of interventricular septal defects of the three types named. The proximity of the conducting mechanism of the heart presents a technical problem in any attempt to repair these defects under direct vision. The majority of the simple interventricular septal defects appear to have a reasonably good prognosis without operation.

In the repair of the third group of the simple anomalies (pulmonary stenosis and interatrial septal defects), reference has already been made to the

reports of two successful series of cases by Swan and Lewis. Swan believes that the results are better with the direct vision approach to pulmonary stenosis than the standard Brock valvotomy. A similar comparison between the open and closed technique of repairing interatrial septal defects will also prove interesting. The time required to correct these defects can be obtained at a fairly safe body temperature level. We feel that something more should be known about the natural life history of interatrial septal defects. Our experience would suggest that young children with interatrial septal defects, who develop failure, are likely to have associated anomalies. So far, we have been using a closed technique in adults.

Our group in Toronto has been rather conservative in applying hypothermia for intracardiac procedures. In cooperation with Dr. John D. Keith and associates of the Hospital for Sick Children, three patients of the first group of complicated anomalies have been operated upon at body temperatures of 20° to 23° C. (68° to 76° F.) with one successful correction of the defect with survival. A patient with tricuspid atresia and one with pulmonary atresia were operated upon by an intracardiac approach without survival. The first patient tolerated interruption of the circulation for twenty minutes with good heart action. We were pleased with the value of hypothermia and the increase in arterial oxygen saturation associated with lower body temperature. The successful case was a total anomalous pulmonary venous drainage corrected surgically by one of us (W. T. M.) by anastomosing the common pulmonary venous trunk to the waist of the left auricle. This entailed partial obstruction of the pulmonary venous drainage for thirteen minutes and total interruption of the circulation for six minutes. The 1-year-old child is remarkably improved.

No interventricular septal defects have been operated upon under direct vision. It is obvious that both this group of cases, and the complicated anomalies will require a fifteen- to thirty-minute period of interruption of the circulation for an adequate correction of the defect. This will entail a body temperature of 20° to 15° C. (68° to 59° F.). There are not only technical difficulties, as mentioned, but these body temperatures are in themselves dangerous.

Two other intracardiac procedures were attempted with fatal results in our series. One was a pulmonary valvotomy under direct vision in an advanced case; the other was an error in the preoperative diagnosis.

Intracardiac surgery under hypothermia for cardiac disease will likely await safer techniques of cooling before this method is generally accepted by all surgeons. It is a stimulating field of investigation, however, since some of these infants and children, such as the cases of transposition of great vessels, are now not only numerous, but also hopeless. Successful correction could result in a relatively normal heart.

Hypothermia as an Adjunct in Poor-Risk Cardiac Surgery.—Fig. 5 represents graphically the principle employed in using hypothermia as an adjunct in current cardiac operations upon poor-risk patients. The body tempera-

ture of 30° C. (86° F.) is safe for both adults and children. At that temperature, judging from experimental evidence, the blood pressure should not be seriously lowered, and the oxygen requirements should be reduced to about 55 to 65 per cent of normal. Our group has operated upon sixteen patients who were very poor risks at body temperatures of 30° C. (86° F.) or slightly lower. About one-half of these were children. The indications for its use in congenital heart disease have been (a) marked cyanosis with an arterial oxygen saturation usually below 68 per cent and (b) cases where partial occlusion of the circulation may be necessary in order to complete the procedure.

In the group of acquired heart disease, hypothermia has been used in those cases where heart failure has not cleared after several weeks of hospitalization on a strict medical regimen. Most of these patients were cyanosed.

Table II lists types of cases that have been operated upon in this group at the Toronto General Hospital and the Hospital for Sick Children.

TABLE II. HYPOTHERMIA—AN ADJUNCT IN POOR-RISK CARDIAC SURGERY
(Patients operated upon)

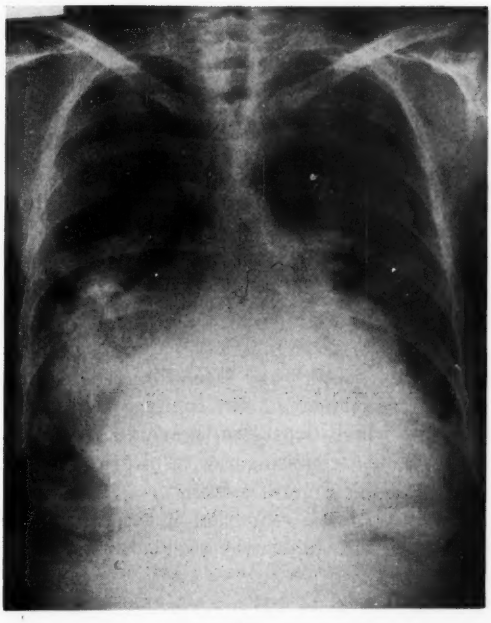
ADULTS		CHILDREN	
Mitral stenosis	7	Tetralogy of Fallot	1
Aortic regurgitation	1	Tricuspid atresia	1
Interatrial septal defects	1	Pulmonary atresia	1
Thoracotomy	1	Transposition of great vessels	3
Body temperatures	24° to 31° C. (75° to 88° F.)		
Total cases	16		
Operative deaths	4		

As indicated by the table, a mortality rate of 25 per cent appears rather high. In addition, one patient with mitral stenosis died in the postoperative period, and two of the patients with congenital heart disease died one and two months, respectively, after operation. They represent a group of cases, however, where operation at normal body temperature would have either been contraindicated, or where a much higher mortality rate than 25 per cent would have been expected.

In the congenital group the procedures carried out were: Blalock operation, pulmonary valvulotomy, and vena caval transplantation for transposition of great vessels. The arterial oxygen saturation rose from an average preoperative level of 69 per cent to an average of 78 per cent after cooling and at the commencement of the operation.

Of the 7 patients with mitral stenosis, 6 were in chronic failure and all had some peripheral and sacral edema. Five were cyanosed, and in those cases where an arterial oxygen saturation was obtained at rest preoperatively, all were below 90 per cent. Four had ascites and pleural effusion which did not clear with medical treatment. They were all emaciated and many had been in chronic failure for several years. Two patients had ceased to respond to diuretics and were in an obvious terminal state. They were treated with cortisone for ten days preoperatively. Fig. 6 shows the chest x-ray of two patients with mitral stenosis operated upon. The patients with aortic regurgitation had

A.



B.

Fig. 6.—*A* and *B*, Chest x-rays of two patients with mitral stenosis operated upon, using hypothermia as an adjunct. The heart size is an index of the type of cases included in the group.

been in chronic failure in bed for four months, and it was felt that hypothermia would allow a longer period of interruption of the aorta and reduce the danger of spinal cord injury.

In the acquired heart disease group, the profound and sustained fall in blood pressure which usually accompanies operation at normal body temperature upon such patients was not encountered. In the majority of these patients the blood pressure was maintained above 80 mm. Hg systolic, with good heart action which tolerated manipulation and showed no tendency to develop ventricular fibrillation.

One cannot conclude anything from such a small series of cases, but it is our firm impression that using hypothermia as an adjunct makes operation possible upon some of these otherwise terminal heart cases. The degree of improvement obtained in such patients with very large hearts has been remarkable. Our opinion of hypothermia as an adjunct in cardiac surgery is supported by the reports of Dogliotti,[23] Bailey,[24] and Muller.[25]

TECHNIQUE

The technique of cooling patients is not as yet standardized in our center. Both cooling blankets and cold-water baths have been used. In adults it is more practical and appeals more to the esthetic sense of the operating room staff to have them cooled with refrigerated blankets. Infants and children may be cooled either way. They are very simply cooled by being placed in a cold-water bath at 6° C. (37° F.). This produces more rapid cooling than the blanket technique. The rate of cooling also depends on their weight. Some infants have cooled at the rate of a degree centigrade every two minutes. The disadvantage in cold-water cooling in infants is the great drift of the body temperature after they have been removed from the cold-water bath, which is a result of the rapid rate of cooling. The use of cooling blankets for children provides better control of the body temperature. In all cooling procedures with an adequate circulation, the drift of body temperature after removal from the cooling medium is dependent upon three factors: (a) rate of cooling, (b) weight of the patient, and (c) temperature and humidity of the operating room.

Our present technique of cooling involves the use of preoperative administration of Phenergan, Largactil,* and Demerol. These drugs may prove to be a great aid in the development of our cooling techniques. Largactil is a ganglion blocking agent which depresses the shivering reflex, and a type of sedative which acts without depressing the respiration. Phenergan and Largactil together potentiate the general anesthetic. Demerol was added for its general sedative effect. With the reduction of the shivering reflex, and body temperature is allowed to fall, under the effect of sedative, provided it is in a cool environment.

Adults have been given 50 to 100 mg. each of Phenergan, Largactil and Demerol intravenously or intramuscularly over a one-hour period before application of the blankets. These drugs are continued intravenously during cooling; at approximately 34° C. (93° F.), anesthesia is induced with a small

*Poulenc Limited.

amount of Pentothal. The patient is intubated with institution of positive pressure artificial respiration with oxygen. Further movement or shivering is controlled with a small amount of Pentothal or ether. The positive pressure respirations are given at about 20 per minute and moderately deep in order to blow off the carbon dioxide and to elevate slightly the pH of the blood. In order to eliminate the deleterious effect of prolonged positive pressure respirations, as previously mentioned, the anesthetic is not induced until the patient has been partially cooled. Fig. 7 shows the present apparatus used in cooling. As mentioned above, our current concept of the control of respirations is a compromise based on evidence that strong positive pressure respirations with oxygen are beneficial since this elevates the pH of the blood, but when carried out for too long it increases venous pressure and predisposes to ventricular fibrillation.

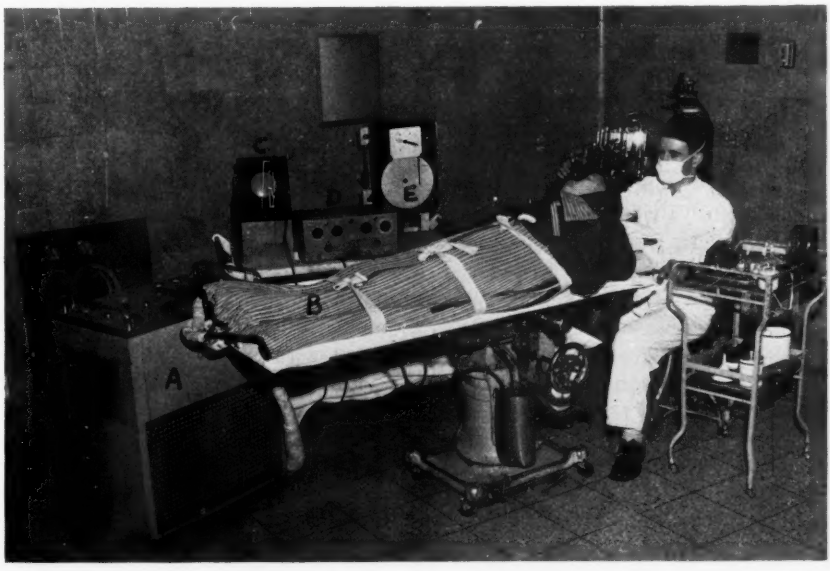


Fig. 7.—Apparatus used to cool patients. *A*, Refrigerator and pump*: *B*, cooling blankets with circulating refrigerant; *C*, cathode ray electrocardiograph; *D*, a combination pacemaker-defibrillator for cardiac resuscitation; *E*, continuous recording rectal thermocouple. (From S. Clin. North America, August, 1954.)

*Thermo-Rite Company Limited, Buffalo, N. Y.

In *children,* the danger of prolonged positive pressure respirations is not significant, since they cool so much more rapidly. These children are prepared with a small dose of Phenergan, Largactil, and Demerol. They are anesthetized with intubation of the trachea before being inserted in the cooling blankets or cold water bath.

At the completion of the operation, the patient is rewarmed to normal body temperature. This may be carried out in three ways: (1) in bed with hot-water bottles, (2) diathermy rewarming, and (3) warm-water bath at 40° C. (104° F.). We have used all three techniques of rewarming. The di-

athermy device[26] rewarms as rapidly as immersion in warm water. On two occasions the temperature drifted to above normal. There have been no other deleterious effects from the diathermy technique. Our present opinion is that at temperatures above 28° C. (83° F.), the patient should be rewarmed by simply placing him in a bed with warm-water bottles. The water bottles should be removed at a body temperature of 33° C. (91° F.). Patients that have been cooled to temperatures below 28° C. (82° F.) should be removed rapidly from this danger zone by either warm water or diathermy rewarming. This should be carried out once again to a body temperature of 33° C. (91° F.) and the rewarming completed with the patient in warm blankets in bed. These techniques will undoubtedly change with more experience.

DISCUSSION

The term "hypothermia" is being used at present to describe any lowering of body temperature. New terms will be required to denote different body temperature levels in order to promote clear thinking. The reduction of the body temperature by a few degrees, originally suggested by McQuiston,[27] to counteract hyperpyrexia during cardiac surgery in children should be clearly differentiated from the lower body temperatures attained at present when hypothermia is used as an adjunct in poor-risk cases.

The new term "artificial hibernation" used by Laborit[28] also requires clarification. He has introduced the use of some very interesting drugs and is developing a new technique of anesthesia. As a result of the depression of the heat regulating center produced by his "lytic cocktail," he has observed a coincident drop in body temperature of only 1° or 2° C.

Hypothermia would appear to be a physiologic approach to some of our problems in cardiac surgery. It is likely that with the current interest and research on this subject, the dangers of lowering body temperature below the present safe levels may be reduced.

SUMMARY AND CONCLUSIONS

1. Some of the physiologic and biochemical changes produced by lowering the body temperature are discussed.

2. The use of hypothermia for intracardiac surgery, at present, has a limited application. Safe body temperatures do not allow a period of interruption of the circulation for long enough to correct the majority of cardiac defects.

3. The use of hypothermia with a body temperature of 30° C. (86° F.) is advised as a useful adjunct in the cardiac surgery of poor-risk cases, both children and adults.

4. Twenty-one cases are discussed. These patients were operated upon at temperatures ranging from 20° to 31° C. (68° to 88° F.).

The cardiology staffs of the Toronto General Hospital and Hospital for Sick Children, Dr. S. M. Campbell and Dr. A. O. C. Smith of the anesthetic services, Dr. D. R. Wilson, Dr. B. G. Grapes, Dr. A. Sirek, Dr. J. W. Rose, and many others cooperated in this work. Dr. R. M. Janes, Professor of Surgery, has sponsored and encouraged the work since its inception.

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DISCUSSION

DR. F. JOHN LEWIS, Minneapolis, Minn.—Dr. Bigelow's excellent work on this subject in the past stimulated us to an interest in hypothermia, and on the basis of our experience I would like to make two points. The first concerns the clinical use of hypothermia to close atrial septal defects under direct vision. Age is no contraindication to

the performance of this operation. In fact, in our own series nine patients over 16 years of age, the oldest of whom was 52 (her defect was successfully closed), have been subjected to this operation. Among the nine there was only one death.

The second point I would like to make concerns the ability of adult animals to tolerate profound hypothermia, body temperatures below 10° C. Dr. Svad Niazi, working in our laboratory, set out to make a study of the mechanism of death due to hypothermia in rats. It was our expectation that these adult rats would die of ventricular fibrillation at about 15° C., as had been reported in the past, but to our amazement we found that with a careful technique of cooling and rewarming they would tolerate much lower temperatures, temperatures previously tolerated, with survival, only by hibernating animals, as Dr. Bigelow has demonstrated. But there has been a striking difference between the way these adult rats took to cooling and the way hibernating animals respond. In contrast to hibernating animals, the adult rats go through a period of complete cardiac arrest at the low body temperatures which may last for a period of two to three hours, with recovery.

I have here an electrocardiogram of a rat being cooled. As the temperature falls below 15° C., the pulse becomes very slow. At about 9° C. cardiac standstill takes place. The artificial respiration is then discontinued and the animal is cooled further to a few degrees above freezing. The animal is rewarmed with hot water. If there is only a thirty- to forty-five-minute period of complete standstill, there are usually no sequelae, although some animals have had neurologic complications. Over three hours of cardiac standstill is not followed by recovery. Apparently post-mortem changes take place even at these low temperatures.

DR. HENRY SWAN, II, Denver, Colo.—I wish to pay tribute to Dr. Bigelow and to bring to your attention one matter of interest concerning the prevention and treatment of ventricular fibrillation in the hypothermic animal. We are talking about the same temperature ranges that Dr. Bigelow is discussing, that is, from 20° to 37° C. Ventricular fibrillation has been the primary bugbear in our laboratory in this temperature range, especially from 25° to 20° C. In the discussion of Dr. Sealy's paper we are all acutely aware of the interest that is going on with regard to potassium in cardiac arrhythmias. Looking on an entirely different aspect of the potassium problem, it was found by Dr. Holmes and Dr. Montgomery in our laboratory that Prostigmin potentiates the flow of potassium across the cell membrane. Working with this clue, we undertook to find the effect of Prostigmin on ventricular fibrillation.

In our laboratory in Colorado if you make a right ventricular incision in the cold animal you will get 100 per cent incidence of ventricular fibrillation. These 23 animals served as controls. All fibrillated. When given 1 c.c. of 1:4,000 Prostigmin intravenously at about 30° C. during the cooling process, ventricular fibrillation occurred in only 7 of 15 animals. When Prostigmin was given to the animals by coronary perfusion (injection directly into the coronary system at the time of occlusion of the circulation) none of the 16 operated on fibrillated.

As you know, Prostigmin is an anticholinesterase; one would presume that if its action was by potentiating the action of acetylcholine, then one could achieve the same effect by giving acetylcholine. This proved to be the case. Of the 5 animals, constantly given acetylcholine through the coronary system, none fibrillated. We made the additional experiment to see if autogenous acetylcholine also produced this result. Continuous stimulation of the distal end of the cut vagus nerve was protective in 4 of 6 animals.

Coronary sinus catheterization is also a stimulus to fibrillation against which Prostigmin was protective. We have had the feeling that this agent was helpful in the last 6 of the 31 patients upon whom we have performed open heart surgery.

DR. C. P. BAILEY, Philadelphia, Pa.—We too were stimulated by Dr. Bigelow's fine basic research and, after a period of animal experimentation in August, 1952, we performed what I believe was the first intracardiac operation under hypothermia. It was four days before the operation performed by Dr. Lewis and Dr. Varco, but was much less

successful because our patient died during surgery. The cause of death was ventricular fibrillation. The condition for which open surgery was carried out was a large interatrial septal defect of the type known as persistent ostium primum.

Our results with hypothermia have not been as good as those presented. We have completely interrupted the circulation in more than 20 cases with a mortality of 66 per cent. Admittedly, we do not do open surgery when we think closed surgery will suffice. These were bad-risk cases such as transposition, extreme tetralogy, and so forth. Nevertheless, the mortality is a little shocking, and one must consider the possible cause. Part of it, I think, is definitely due to the hypothermia which depresses myocardial contractability. We feel that acquired heart disease in adults is so poorly amenable to this modality as to contraindicate application. Second, even in the congenital types, there is a great tendency to the development of ventricular fibrillation which may be irreversible. The third point is related to the open cardiac surgery. We firmly believe that one cannot safely operate on the left side of the heart by an open technique because of the high risk of coronary embolization. When a large septal defect is present, opening the right side of the heart is really the same as opening the left side of the heart, and, although the risk is not so great as when the left side is opened directly, there is, nevertheless, a residual risk from that source. I know that Dr. Geoghegan and Dr. Lam have a way of getting air out of the coronaries, but in our hands it has not been too successful clinically. We think coronary air embolism should be avoided if possible.

We feel that, at the moment, Potts and McQuiston[27] have stated the situation appropriately. Probably the best immediate place for hypothermia is as an adjunct to the orthodox treatment of cyanotic heart disease. Preferably it should not be a very severe level of hypothermia. In fact, Potts has expressed the view that the more important element may be the avoidance of hyperpyrexia during and after surgery rather than the actual lowering of body temperature.

DR. EDWARD E. AVERY, Chicago, Ill.—Dr. Bigelow mentioned that hyperventilation prevented some of the adverse effects leading to ventricular fibrillation during hypothermia, but that prolonged positive pressure anesthesia also produces deleterious effects on the heart and circulation. The injurious effects of prolonged positive pressure breathing has been reported by Cournand, Motley, and Werko in 1947. More recent work by Mörch and Maloney has shown it is the mean pressure that is important and that the positive pressure phase should be relatively short, with a gradual peak and rapid fall of pressure to atmospheric or subatmospheric levels. Inspiratory pressures should occupy not more than one-half of the respiratory cycle, and the expiratory phase less than one-half of this time. Mörch and Benson have shown by this method of ventilation that pulmonary ventilation and cardiac output are enhanced. Mörch, in 2,000 cases of positive-negative anesthesia, has proved its value in adequate ventilation of the lungs, prevention of hypercarbia, reduction in amount of anesthetic agent used per unit of time, and under experimental conditions has demonstrated actual partial maintenance of circulation by this method alone. Although Ankeny at Western Reserve has shown that positive and negative pressure anesthesia does not increase the circulation in the exposed lung during open thoracotomy, Thompson and Rockney have shown that positive-negative ventilation will benefit the circulation of the contralateral lung, improve pulmonary ventilation, and prevent hypercarbia and ventricular fibrillation. It is almost impossible to maintain adequate positive pressure anesthesia by hand because of the difficulty in maintaining proper rhythmic pressure changes with exact mean pressures and proper pressure profiles over long periods of time. A mechanical respirator is needed.

DR. RALPH A. DETERLING, New York, N. Y.—We too wish to pay tribute to Dr. Bigelow for his great pioneering work in this field. We have a few comments to make which I think should be re-emphasized, although I think some of the discussants have mentioned them. In the last year and a half we have carried on a joint program with our anesthesia department and, unlike some groups, we have studied hypothermia in dogs, with

no surgery being performed. Many of the findings we have accumulated during this time do not completely agree with some of the basic data which have been reported and which form the basis for certain specific recommendations as to management of patients. I think that clarification has to be made, because if one has an animal or a patient with an open thoracotomy and a cardiac surgical procedure, there are certainly introduced additional factors beyond those resulting from hypothermia. Caval occlusion has been a part of some of the procedures, which in itself produces certain changes.

We have operated on eight patients under hypothermia, about half of them brought down to 23° C. and half to about 31° C. The mortality was about 30 per cent. The second point I wish to emphasize is that I do not think hypothermia played a major role in the mortality, since the patients who did not survive were those who had large septum primum, transposition, or a condition of a serious nature.

Last week we had a sad experience with an adult with an arterial saturation of 18 per cent from a tetralogy of the infundibular type. We had planned to keep him at 30° C. but he drifted unpredictably down to 25° C. At that point he fibrillated, as we were opening the chest. We tried electric shock several times and even potassium chloride, with no success. In addition, we performed cardiac massage for a considerable time. I think Dr. Ralph Adams is quite right in feeling that the fibrillation problem is not yet a simple one.

In the older adult group with abdominal aortic aneurysms, I have preferred to try the Laborit drug hibernation technique. I have used it on three old high-risk patients with satisfactory results. I was afraid to use surface hypothermia in that group, although I understand some surgeons have done so.

DR. W. G. BIGELOW (Closing).—I should like to thank the discussants very much. Dr. Lewis made a remark about cooling a rat down to very low temperatures with cessation of cardiac function for long periods, and rewarming. This is a function of the laboratory animal which he has used. He is using an animal which is small and can be rapidly cooled and rapidly rewarmed. This experiment is difficult to duplicate on an animal of larger body mass, because its body temperature cannot be lowered rapidly by ordinary surface cooling techniques. In the time that it takes to get them into the low temperature zone, anoxia due to cessation of cardiac function would intervene and put the heart in a state where it could not be resuscitated. It is an interesting experiment. This is possible in larger animals such as the groundhog. One may cool them to 3° C. and interrupt their circulation with the heart open for one to two hours, with survival. In these animals, however, the heart continues to function, and circulation is maintained.

Dr. Swan's use of Prostigmin is very interesting. We have been rather parallel in our thinking, both regarding the effect of hyperventilation and now the significance of the acetylcholine mechanism which has caught our interest. I agree with Dr. Bailey that the present most useful place for hypothermia is as an adjunct. I might make a comment on Dr. Deterling's problem of drifting of the body temperature after removal from the cooling medium. There are two methods by which you may cool an individual, with blankets or with a cold-water bath. In adults we like to use blankets and, if the blanket is good and cold, with the help of ice bags one can cool patients fairly rapidly. It is somewhat more esthetic to the operating room staff to have the adults in blankets. However, in children both blankets and cold-water baths are acceptable. The advantage of cold water is that it cools them more quickly. The disadvantage is that they drift more and it is difficult to control the temperature once you take them out of the bath. The use of blankets in children is acceptable because it gives a more constant temperature without the marked tendency to drift.

THE SURGERY OF INFUNDIBULAR PULMONIC STENOSIS WITH INTACT VENTRICULAR SEPTUM (A TYPE OF "PURE" PULMONIC STENOSIS)

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PULMONIC stenosis in the presence of an intact ventricular septum is perhaps the most gratifying and satisfactory type of intracardiac lesion with which the present-day cardiovascular surgeon must deal. For this state of affairs the profession is indebted to the initial efforts of Doyen[1] in 1913 and, more recently, to Sellors[2] and Brock[3] who were instrumental in the development of the direct surgical approach for the relief of this condition. The past five years have witnessed widespread interest in the problem of structural obstructions within the outflow tract of the right ventricle.

Several investigators[4-8] have reported that pulmonic stenosis is usually valvular in type when the interventricular septum is intact, whereas an infundibular type of stenosis is common in the presence of an interventricular septal defect. The purpose of this communication is to present the findings in six patients with pulmonic stenosis of the infundibular type, but in which the ventricular septum was intact. In five patients the infundibular location of the stenosis was suggested preoperatively by roentgenographic means and by cardiac catheterization; in all six patients the diagnosis was confirmed at surgery.

For the sake of simplicity, hereafter when pulmonic stenosis (valvular, infundibular, or combined) occurs in the presence of an intact ventricular and atrial septum, it will be referred to as pure pulmonic stenosis. If pulmonic stenosis occurs with an intact ventricular septum but with an interatrial communication (patent foramen ovale or true atrial septal defect) it will be referred to as the trilogy of Fallot.[9]

The signs and symptoms common to these two entities, that is, pure pulmonic stenosis and the trilogy of Fallot, are related to an increased pressure in the right ventricle and often to a decreased pulmonary blood flow. Increased pressure in the right ventricle occurs in association with pulmonic stenosis with or without an intact ventricular septum, and the pressure in the

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pulmonary artery is always lower than that in the right ventricle. Increased right ventricular pressure may also occur in any anomaly characterized by a large left-to-right shunt or in idiopathic pulmonary hypertension, but in these conditions the pressure in the pulmonary artery is also elevated.

Inadequate pulmonary blood flow may result from failure of one or both venae cavae to enter the right atrium or from anatomic obstruction at different levels in the right side of the heart and/or in the pulmonary arterial tree, for example, tricuspid atresia, Ebstein's disease, pulmonary infundibular or valvular stenosis, pulmonary atresia, or in severe pulmonary hypertension.

There is no known natural mechanism to relieve the high pressure in the right ventricle, although surgical procedures may afford some relief for the elevated pressure in some types of malformation. Furthermore, although persistent patency of the ductus arteriosus and the establishment of a collateral circulation may partially compensate for an inadequate pulmonary blood flow, the former usually affords only temporary and often an incomplete blood supply, and collateral circulation is often insufficient to maintain an adequate pulmonary blood flow. The establishment of an adequate pulmonary blood flow has been made possible in a number of these conditions by different surgical procedures, such as systemic-pulmonary artery anastomosis,[10, 11] valvotomy,[12, 13] and infundibular resection.[14-17] In others no surgical amelioration has been feasible to date.

Frequency.—Pulmonic stenosis as part of the tetralogy of Fallot is one of the most frequent malformations of the heart[4] and is certainly the commonest lesion in the cyanotic group of congenital heart diseases. Pure pulmonic stenosis and trilogy of Fallot have until recently been considered very rare malformations of the heart. In Fallot's[9] series of 40 cases of cyanotic congenital heart disease, 7 were examples of the trilogy, that is, pulmonic stenosis with an intact ventricular septum, interatrial communication, and right ventricular hypertrophy. In Abbott's[18] series of autopsies there were 25 instances of pulmonic stenosis with an intact ventricular septum in 1,000 cases of congenital heart diseases; of these, 16 had a patent foramen ovale, and in the remainder the interatrial septum was anatomically closed. In Escalle's[19] series there were 16 instances of pure pulmonic stenosis as compared to 21 of the trilogy of Fallot. Sir Arthur Keith[20] in 1909 was probably the first author to call attention to the occurrence of pulmonic infundibular stenosis with an intact ventricular septum. In 1949, Greene and associates,[21] reviewing the literature dealing with pulmonic stenosis with an intact ventricular septum, found 17 instances of infundibular stenosis in a total of 68 cases; in 5 of these there was also an associated valvular stenosis. Selzer and co-workers,[22] in a study including 23 selected cases of pure pulmonic stenosis, found 5 instances of myocardial infundibular stenosis, 2 of membranous infundibular stenosis, and 15 of valvular stenosis; of 29 cases of the trilogy of Fallot, there was only one instance of "subvalvular membrane," the stenosis being valvular in the remaining 28 patients. In a series of 25 cases of pulmonic stenosis with intact ventricular septa operated on by the authors, there were 6 instances in which the stenosis was infundibular or combined valvular and infundibular.

Diagnosis of Pulmonic Stenosis With Intact Ventricular Septum.—An increasing amount of clinical,[21, 22, 23, 24, 33, 37, 39] electrocardiographic,[31] roentgenologic,[32, 34] angiocardiographic,[32, 34] physiologic,[21, 23, 25, 27, 29, 30, 35, 36] surgical,[2, 3, 5, 12, 16, 23, 30, 39, 41] and anatomic[34] data concerning this malformation have been accumulated in recent years. The correlation of these data has made possible a rather accurate clinical diagnosis of pulmonic stenosis with intact ventricular septum. However, to date a *definitive* diagnosis of this malformation can be made only by interpreting the results of cardiac catheterization.

The symptoms, signs, and physiopathologic findings in this entity may vary somewhat with the degree, site, and type of stenosis as well as with the presence or absence of an interatrial communication. The usual manifestations in infants and children with pulmonic stenosis with an intact ventricular and atrial septum will be presented. The changes brought about by the presence of an associated interatrial communication and the differential diagnosis of infundibular and valvular stenosis will be discussed.

Pure Pulmonic Stenosis.—Dyspnea, usually noted on exertion and out of proportion to the degree of cyanosis, is the most frequent symptom. Easy fatigability is noted in association with dyspnea. Inadequate gain in weight is a common complaint. Squatting, although not as frequent as in tetralogy of Fallot, may occur.

Physical examination usually reveals no cyanosis; when present it seems to be due to severe peripheral venous unsaturation (peripheral cyanosis) and not to a venous-arterial shunt, as the arterial oxygen saturation is always normal. The chest may be symmetrical or may show varying degrees of precordial bulge as a result of right ventricular hypertrophy, but cardiac enlargement is usually not detected clinically. Auscultation reveals a systolic murmmur, often accompanied by a systolic thrill, and located at the second left interspace. Diastolic murmurs are extremely rare. The second sound in the pulmonary area is often clear and diminished in intensity, but a normal or loud P_2 may be noted. The liver is usually neither enlarged nor pulsating, but in the more severe forms or advanced stages an enlarged and pulsating liver may be noted. The systemic systolic pressure is usually at or below 100 mm. Hg and the pulse pressure is narrow.

The values for hemoglobin, red cells, and hematocrit are usually normal and the circulation time is normal or slightly prolonged.

The electrocardiographic tracings reveal right axis deviation, right ventricular hypertrophy, and often tall, peaked P waves, especially in limb Lead II, suggesting right atrial enlargement or strain. Changes in the T waves indicative of myocardial strain may be noted in the more severe and advanced cases.

Fluoroscopic examination reveals the heart to be normal or moderately enlarged. The apex is often elevated and the pulmonary conus convex. The pulmonary artery and its main branches at the hilus are usually large but quiet; however, a "jet pulsation" into the left pulmonary artery may be seen

at times. The lung fields themselves are clear. Right ventricular enlargement is suggested in the frontal view and is confirmed by rotating the patient in the oblique positions. In contrast to the tetralogy, the pulmonary window is not clear. Roentgenograms confirm the fluoroscopic findings and demonstrate decreased vascular markings in the outer two-thirds of the lung fields.

Cardiac catheterization reveals that pressures in the right atrium are usually elevated. The systolic pressure in the right ventricle is always elevated and seems to be directly related to the degree of stenosis; it may be lower, equal to, or higher than the systemic systolic pressure. The occurrence of a pressure in the right ventricle which is significantly higher than the systemic systolic pressure is, for practical purposes, diagnostic of pulmonic stenosis with an intact ventricular septum. On the other hand, in all malformations having a large ventricular septal defect, the systolic pressure in the right ventricle usually approximates the systemic systolic pressure. The pressures in the pulmonary artery as recorded by conventional means may be normal, but are usually decreased. If the internal diameter of the stenosis (as described in the surgical and anatomic protocols) is compared to the external diameter of the catheters used, the validity of the pressure recordings beyond the stenosis, where measured by such conventional means, may be questioned; it seems reasonable to assume that, at least in the more severe stenoses, the passage of the catheter through the narrowed area would add an extra "stenosing" factor and would thus contribute to the decreased pressure recorded in the pulmonary artery. If this is true, the pressures recorded directly from the pulmonary artery (not via the right ventricle) would be expected to be higher than those recorded by the conventional route, again in the more severe forms of stenosis. That varying degrees of pulmonary hypertension may be found in association with pulmonary stenosis has been verified in rare instances.[11, 22, 29] The importance of the recognition of such occurrence is obvious when one contemplates surgery for the purpose of increasing pulmonary blood flow since the surgical procedure would then add a potentially hypertensive factor to an already existing pulmonary hypertension.

The oxygen studies performed on samples of blood obtained at cardiac catheterization reveal no evidence of intracardiac shunts. Measurement of the pulmonary capillary blood flow may reveal evidence of an extracardiac arteriovenous shunt if a collateral circulation has been established between the bronchial and the pulmonary arteries, and thus the total pulmonary blood flow will exceed the systemic blood flow. In the absence of shunts the pulmonary and systemic blood flows are equal and usually decreased, but may be normal or rarely increased. Soulié and associates[25] have demonstrated normal or increased pulmonary blood flow in association with severe pulmonary stenosis.

Angiocardiography reveals that the dye follows a normal course. The significant findings which, however, are not constant, are the retention of dye in the right ventricle, the visualization of the poststenotic pulmonary artery

dilatation, and the decreased vascular markings in the outer two-thirds of the lung fields. These, however, are only indirect diagnostic signs of pulmonary stenosis; the stenotic area can be directly visualized roentgenographically only by special angiocardiographic technique employing the direct injection of the dye into the entrance of the outflow tract of the right ventricle. Special apparatus must be used in order to obtain the necessary films which will demonstrate both the site and the degree of stenosis.

Trilogy of Fallot.—The trilogy of Fallot may be indistinguishable from pure pulmonic stenosis if the right-to-left shunt at the atrial level is insignificant. With larger shunts, however, cyanosis, although it may be absent early, is usually present. It is first noted on exertion and progresses slowly. The cyanosis is usually mild to moderately intense and is predominantly central in origin, being the result of a venous-arterial shunt at the atrial level; peripheral venous unsaturation may contribute to its intensity. Associated with the cyanosis are polycythemia and, at a later date, clubbing of the fingers and toes. In the presence of a large right-to-left shunt there is a decrease in the circulation time. The pressures in the right atrium are almost always elevated and the shunt is from right to left. In some instances a bidirectional shunt at the atrial level may occur, but it is predominantly from right to left. If the diagnosis of pulmonic stenosis with an intact ventricular septum has been established, the passage of the catheter through an interatrial communication differentiates a pure pulmonic stenosis from a trilogy of Fallot. In the presence of a large right-to-left shunt angiocardiography will initially reveal rather poor opacification of the left atrium, ventricle, and aorta followed later by reopacification of these chambers and aorta, and the systemic blood flow will exceed the pulmonary blood flow.

Differential Diagnosis of Valvular and Infundibular Stenosis.—At the present time valvular stenosis cannot be positively differentiated clinically from infundibular or combined stenosis. In the tetralogy of Fallot, where the stenosis is predominantly infundibular, the murmur is usually located lower along the left sternal border in the third to fourth interspace. On the other hand, in pulmonic stenosis with intact ventricular septum, where the stenosis is usually valvular in type, the murmur is loudest in the second left interspace. More clinical data will be necessary to determine whether the same correlation will be found between the location of the murmur and the site of the stenosis when the ventricular septum is intact.

The presence of a split second sound at the base tends to exclude the diagnosis of pulmonic stenosis. Whenever this finding is present in association with the other findings of pulmonic stenosis with an intact ventricular septum, it is suggestive evidence of an infundibular stenosis with a normal pulmonary valve.

Fullness or convexity of the pulmonary artery segment suggests the presence of a large poststenotic dilatation of the pulmonary artery and thus favors a diagnosis of a valvular type of stenosis (Fig. 1). Straightness or

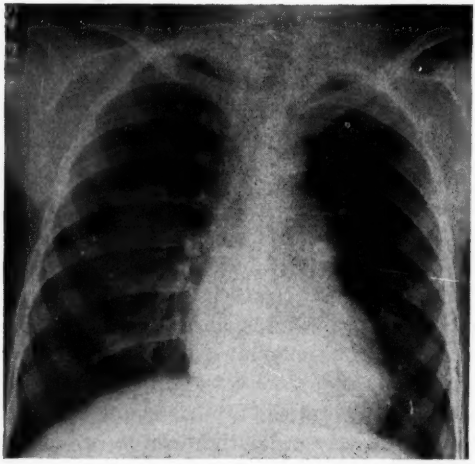
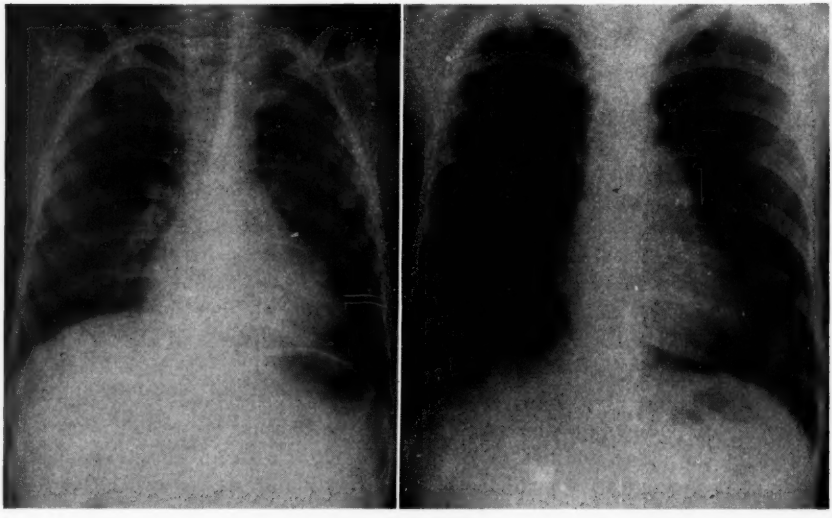


Fig. 1.—Typical posteroanterior roentgenogram of pure valvular pulmonic stenosis. Note the fullness and convexity of the pulmonary artery segment on the upper left border of the cardiac silhouette produced by poststenotic dilatation.



A. *B.*

Fig. 2.—Posteroanterior roentgenograms of pure infundibular pulmonic stenosis. Although there is usually a concavity in the region of the pulmonary artery segment as in *A* (Case 4), there may be a convexity as in *B* (Case 1), which may confuse the diagnosis on purely radiographic grounds.

concavity of the pulmonary artery segment, on the contrary, suggests the presence of a normal or hypoplastic pulmonary artery with an infundibular rather than a valvular stenosis (Fig. 2, *A*). The presence of a right aortic arch tends to produce a straight or full shadow in the region of the pulmonary artery segment, and its occurrence must be considered when evaluating this segment.

The continuous recording of pressures while withdrawing the catheter from the pulmonary artery under fluoroscopic guidance is the best available method of differentiating the various types of stenosis. In *pulmonary valvular stenosis* there is a sharp rise of the systolic pressure and a fall of the diastolic pressure to zero or negative values at the level of the pulmonary valve (Fig. 3). In *infundibular stenosis* the systolic pressure is approximately the same

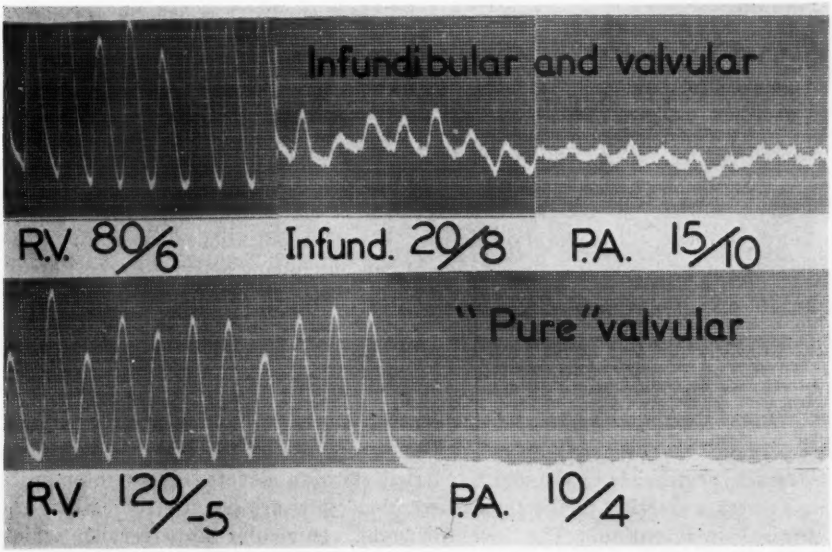


Fig. 3.—Continuous manometer tracings (defective intervening portions removed) as the cardiac catheter is withdrawn from the pulmonary artery into the right ventricle. In the upper tracing the high right ventricular pressure has been reduced in the infundibulum by the intervening stenosis. The existence of a second stenosis at the pulmonary valve is responsible for a further reduction. The lower tracing depicts the typical pressure change from pulmonary artery to right ventricle seen in pure valvular stenosis.

in the pulmonary artery and in the infundibulum distal to the stenosis; proximal to the stenosis, however, there is a sharp rise in the systolic pressure; the diastolic pressure falls at the level of the pulmonary valve and remains constant in the right ventricle proximal and distal to the stenosis. In *combined valvular and infundibular stenosis* there is a rise in the systolic pressure in the region between the valvular and the infundibular stenosis, followed by a second rise proximal to the infundibular stenosis (Fig. 3); the diastolic pressure falls proximal to the valvular stenosis and is then of the same magnitude in the right ventricle proximal and distal to the infundibular stenosis.

At the time of surgery the site and degree of stenosis can be ascertained by both extra- and intracardiac exploration. In valvular stenosis varying degrees of poststenotic dilatation are usually noted in the pulmonary artery and a systolic thrill is palpable distal to the valve; by digital invagination of the pulmonary artery the orifice of the pulmonary valve can be obliterated with resultant disappearance of the thrill. The pulmonary valve can be palpated as a stiff, dome-shaped structure; a high and short infundibular stenosis located immediately subjacent to the valve may simulate these findings. In the typical infundibular stenosis, however, the thrill is best felt in the wall of the outflow tract of the right ventricle, distal to the site of the stenosis, although it may extend into the pulmonary artery. A short infundibular stenosis of the diaphragmatic type (membranous infundibular stenosis) produces a poststenotic, thin-walled ventricular chamber. The more common, long, narrow infundibular stenosis (myocardial infundibular stenosis) is usually associated with hypertrophy of the outflow tract. Combined infundibular and valvular stenoses are frequently associated with a hypoplastic pulmonary artery.

The value of pressure recordings at surgery for the localization of the primary obstruction and for the demonstration of other obstructions has been pointed out by Kirklin and associates.[23]

The functional significance of pulmonic stenosis, especially of the infundibular type, is better appreciated by cardiac catheterization, selective angiocardiography, and surgical exploration than by direct visualization at necropsy. The striking difference in the appearance of the pulmonary arteries at the time of surgery and at post-mortem examination in patients with tetralogy of Fallot has been stressed by Soulié; in like manner the stenosis as well as the poststenotic dilatation in patients with pulmonic stenosis with an intact ventricular septum may be more striking in vivo than at the autopsy table.

Indications for Surgery.—No definite criteria for the surgical treatment of pulmonic stenosis with an intact ventricular septum have been established. Severe hypertension in the right ventricle with an elevated diastolic pressure or a great reduction in the pulmonary blood flow are definite indications for surgical intervention. The level of right ventricular hypertension which should serve as an indication for surgical correction varies with different authors from 50 to 100 mm. Hg systolic. Systolic pressures above 100 mm. Hg are generally accepted as indicating the need for surgical intervention. Polycythemia, stunting of growth, electrocardiographic evidence of severe right ventricular hypertrophy, and myocardial strain indicate the need for surgical treatment. From the experience of several writers it appears that the age of the patient must be considered in evaluating the indications for surgery, the higher mortality figures occurring among those patients under 2 years and beyond 20 years of age. In general this same variation in surgical mortality rates as related to age applies to all forms of congenital heart diseases.

Surgical Technique.—Although the direct and the indirect approaches have been successfully used to increase pulmonary blood flow in patients with the tetralogy of Fallot, only the direct approach should be used in patients with pulmonic stenosis with an intact ventricular septum. In the few in-

stances in which systemic-pulmonary artery anastomosis has been used[13, 14, 33] for this condition, the operation was invariably followed by right-sided failure. Relief of the strain on the right side of the heart is the primary goal and an increase of pulmonary blood flow follows. According to the type of stenosis, therefore, valvotomy and/or infundibular resection must be used.

The technique as used by the authors has been presented in detail in previous communications.[16, 39, 41] Certain modifications based on a greater experience should be stressed. A rectangular flap of pericardium is raised medially after access to the heart has been obtained through a left parabolic submammary incision entering the pleural space through the third anterior interspace. The entire outflow tract of the right ventricle and the roots of the great vessels are thus exposed. Adequate time must be taken for careful inspection and palpation of these structures, as numerous variations in anatomic detail may at first be confusing. As a rule, the infundibular chamber will consist of a muscular wall similar to, but a little thinner than, the right ventricular myocardium proximal to it. Frequently the wall will be membranous, and similar in consistency and texture to the structure of the pulmonary artery. In this instance it may be difficult to differentiate immediately between the chamber and the artery, but further inspection and exploratory intracardiac probing should clarify the situation. That infundibular stenosis is present is detected by a systolic thrill proximal to the pulmonary valve at the root of the pulmonary artery. The absence of poststenotic dilatation of the pulmonary artery and the inability to delineate a fused pulmonary valve cone with its jetlike systolic flow when digitally invaginating the flaccid arterial wall lend further evidence that the stenosis is infundibular rather than valvular.

Of greatest importance is the accurate localization of the level of maximal infundibular obstruction, for it is directly over this point that the transventricular incision should be made when employing this technique. This area can be recognized in a number of ways. It is the point where the systolic thrill is rather abruptly lost when passing the finger along the wall of the chamber away from the pulmonary artery. In addition, numerous twiglike coronary veins can often be seen to encircle the lower border of the infundibular chamber swinging out from the more perpendicularly placed larger coronary veins running over the interventricular septum. Most accurate localization is obtained by intraventricular exploration first with a small malleable probe, next with a small urethral sound, and finally with the long-shafted resecting instrument* itself.

Each of these instruments in turn is gently passed through the myocardium, the two probes without an incision and the infundibular resecting rongeur through a 1 cm. incision. Each is passed upward through the infundibulum and into the pulmonary artery where it can be readily palpated. They may be rotated in the valve area to determine the patency of the valve. They are then directed backward to the floor of the artery and gradually withdrawn by dragging along the floor of the infundibular chamber, which

*Modified pituitary forceps made by George P. Pilling & Son Co., Philadelphia, Pa.

may vary from a peanut to a lemon in size. As the infundibular ridge or crest is approached, the instrument will ride up over it only to fall off downward into the large lumen of the right ventricle proper. By rocking the instrument gently back and forth over the hypertrophied muscular or membranous crest very accurate localization can be obtained. The jaws of the resector can then be opened, the full thickness of the crest engaged, and a generous bite removed within the mouth of the instrument (Fig. 4). This may

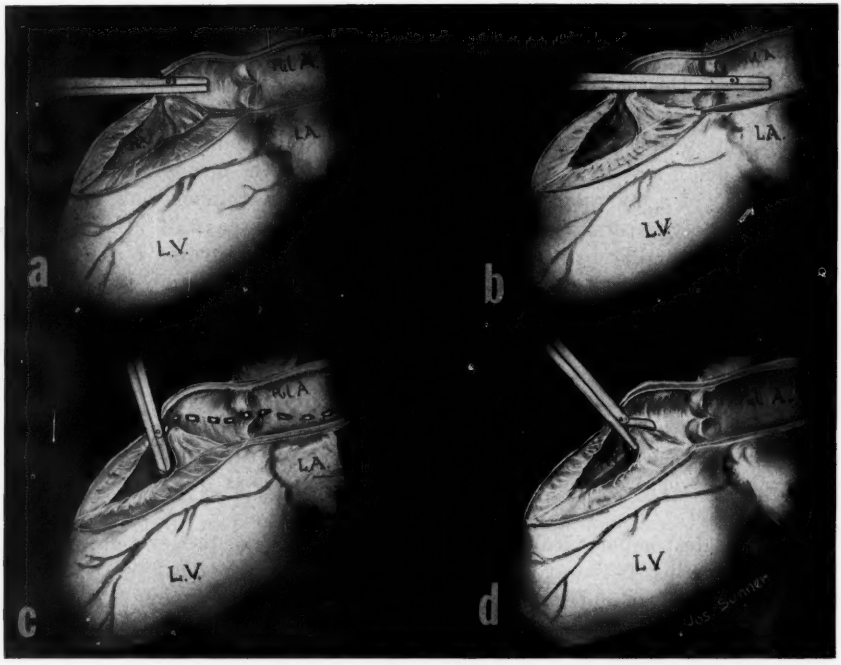


Fig. 4.—The authors' method of infundibular resection. *a*, The resecting instrument is inserted through the myocardium directly over the infundibular obstruction. *b*, The instrument is passed into the pulmonary artery where the status of the valve can be palpably determined. The jaws of the instrument can be opened at this level to further evaluate the valve area. *c*, The instrument is slowly withdrawn in contact with the floor of the outflow tract. It rides over the obstruction and falls into the right ventricle, providing accurate localization; *d*, The jaws are opened and a portion of the obstructing ridge resected. This can be repeated until most of the ridge has been removed.

be repeated three or four times until an entirely adequate channel has been fashioned, after which the infundibular chamber will swell and become tense. The same increased pulmonary flow and pressure will be noted within the pulmonary artery and the thrill, previously soft and localized, will usually become coarse and generalized. If facilities are available, it is advantageous to measure directly the pressures within the ventricle, chamber, and artery, for in this way the completeness of the resection can more readily be determined. A most adequate result can be obtained, however, without these refinements, because sight and the sense of touch are usually sufficient.

With reasonable care one need have little fear during such a resection, for the ventricular septum is intact and no vital structures can be encountered and accidentally injured. When employing this technique in the tetralogy of Fallot, in which there is a large interventricular septal defect lying just beneath the infundibular ridge, the resector must be directed away from the aorta toward the septum to avoid the anterior cusp of the aortic valve which may recoil into the opening of the septal defect. It is wise under these circumstances to time the resecting bite in ventricular systole at a time when the valve leaflets are flattened against the aortic wall. Bleeding from the myocardium is easily controlled between bites by digital pressure or by crossing the initially placed stay sutures. Two or three interrupted sutures of fine cotton are sufficient to close the small myocardial wound.

Postoperative Changes.—Following a successful valvotomy or infundibular resection, the portion distal to the stenosis will be full, tense, and a conspicuous resistance to compression is palpable. The tolerance to activity increases, and cyanosis, if present, usually disappears at or shortly after surgery. The murmur and thrill may change in intensity, become more diffuse or occasionally disappear; some murmur, however, persists. Some degree of increased cardiac enlargement (radiographic silhouette) often occurs shortly after operation and persists for variable periods of time. This may be evidence of a successful operation since cardiac enlargement may be the result of decompensation secondary to increased cardiac output. The pulmonary vessels usually appear more conspicuous and pulsate more vigorously than before the operation. The electrocardiographic tracings frequently reveal inversion of the T waves, these returning to normal after a period of a few months. A longer follow-up is necessary to determine the rapidity of the regression of the electrocardiographic evidence of right ventricular hypertrophy. More postoperative physiologic studies are necessary before the course of the hemodynamic changes can be predicted. While some have demonstrated a significant decrease of pressure in the right atrium and ventricle,[23, 25] others have found no significant changes.[35] Some increase of the pulmonary blood flow occurs and the peripheral arterial oxygen saturation, if previously low, rises to normal or near normal levels. From the work of the group at the Mayo Clinic it seems probable that the hemodynamic changes are directly related to the degree of normalcy that can be achieved by surgery. To achieve normalcy in pulmonic stenosis with an intact ventricular septum, as much of the obstruction should be removed as possible. This probably is not true when the same surgical technique (the direct approach) is used for pulmonic stenosis in the presence of a ventricular septal defect. Here, less extensive surgery is probably desirable, as it results in adequate pulmonary blood flow without producing pulmonary hypertension. Furthermore, irrespective of the degree of surgical resection or valvular incision, the pressure in the right ventricle in these conditions will remain practically unchanged. Although considerable emphasis has been placed on the prognostic importance of postoperative physiologic studies, long, regular clinical follow-up cannot and should not be replaced as the best method of appraisal of the results of surgery.

Mortality.—The patient with pulmonic stenosis with an intact ventricular septum usually progresses from an asymptomatic period to a period of increased symptoms, followed by rapid deterioration terminating in right-sided cardiac failure. Prior to surgery the life expectancy averaged 25 to 30 years.[34]

In our experience the mortality from direct intracardiac valvotomy and infundibular resection varies considerably with the type of malformation for which they are used. Whereas in tetralogy of Fallot and in tetralogy-like malformations the over-all mortality has been 20 per cent in a total of 60 cases operated upon (less than 10 per cent in the last 25 cases), in pulmonic stenosis with an intact ventricular septum there have been no deaths in a total of 25 cases treated (19 valvular, 4 infundibular, and 2 both valvular and infundibular).

CASE REPORTS

CASE 1.—B. T., a 9-year-old white girl, was admitted to St. Chistopher's Hospital for Children because of exertional dyspnea, easy fatigability, and "squatting" since the age of 4 years. A cardiac murmur had been first noted at the age of 10 days.

Physical examination revealed a thin but well-developed girl with slightly dusky-appearing nail beds. Blood pressure was 102/72. There was a harsh Grade 4 systolic murmur and a thrill present in the second and third left interspaces. P_2 was diminished. (Table I.)

The hemoglobin at the time of admission was 14.3 Gm., erythrocytes 4.62 millions per cubic millimeter. The hematocrit was 51. Her circulation time (arm to tongue) was 9 sec-

TABLE I. CLINICAL MANIFESTATIONS AND ELECTROCARDIOGRAPHIC FINDINGS IN PULMONIC STENOSIS (INFUNDIBULAR)

	AGE (YR.)	DYSPNEA	CYANOSIS	SQUATTING	MURMUR	THRILL	P_2	POLYCYTHEMIA	ELECTROCARDIOGRAM
Case 1 B. T. Female	9	On exertion		Yes	Systolic, II-III	Systolic, II-III	Diminished		RAD RVH
Case 2 M. L. Male	5	On exertion			Systolic, III	Systolic, L. S. B.	Diminished		Peaked P_2 RAD RVH
Case 3 J. B. Female	13	On exertion		Yes	Systolic, left base	Systolic, left base	Not split diminished	Mild	Peaked P_2 RAD RVH
Case 4 T. M. Male	6				Systolic, III	Systolic, II-IV	Diminished		Extrasystoles RAD RVH
Case 5 R. B. Female	6	On exertion	Moderate		Systolic, II-III	Systolic, L. S. B.	Diminished	Mild to moderate	Peaked P_2 RAD RVH
Case 6 G. H. Female	3	On exertion		Yes	Systolic, II-III	Systolic, II-III	Diminished, clear	Mild to moderate	RAD RVH

II-III = Second and third interspaces.
RAD = Right axis deviation.
RVH = Right ventricular hypertrophy.
L.S.B. = Left sternal border.

onds. Electrocardiographic tracings revealed right axis deviation and right ventricular hypertrophy. Roentgenograms and fluoroscopic examination of the chest revealed the heart to be of normal size and contour; the pulmonary artery segment was slightly convex.

The preoperative results of cardiac catheterization and of angiocardiography are listed in Tables II and III.

TABLE II. RESULTS OF CARDIAC CATHETERIZATION IN PATIENTS WITH PULMONIC STENOSIS (INFUNDIBULAR)

(Preoperative)

	OXYGEN (VOL. PER CENT)						PRESSURES (MM. HG)			
	S. V. C.	I. V. C.	R. A.	R. V.	P. A.	F. A.	R. V.	INF.	P. A.	B. A.
Case 1, B. T., Female, Aged 9	13.9	—	13.9	13.8	—	87%	40/0	—	—	102/72
Case 2, M. L., Male, Aged 5	12.2	13.7	12.2	11.3	12.6	92.8%	75/0	40/0	26/15	100/80
Case 3, J. B., Female, Aged 13	11.6	—	11.3	10.3	—	87.9%	134/0	—	—	100/74
Case 4, T. M., Male, Aged 6	8.1	—	8.2	8.1	8.2	93%	150/0	28/0	20/12	90/60
Case 5, R. B., Female, Aged 6	13.9	—	14.3	14.6	13.3	84%	120/–5	—	10/4	100/70
Case 6, G. H., Female, Aged 3	11.1	13.02	11.0	11.6	—	87%	100/–5	55/0 80/0	—	86/60

S.V.C. = Superior vena cava.
I.V.C. = Inferior vena cava.
R.A. = Right atrium.
R.V. = Right ventricle.
P.A. = Pulmonary artery.
F.A. = Femoral artery.
INF. = Infundibular chamber.
B.A. = Brachial artery.

Surgery was performed on Feb. 25, 1951; the preoperative diagnosis was pulmonic stenosis with an intact ventricular septum. At operation the thrill was maximum in the thin-walled, dilated distal portion of the infundibular chamber (Fig. 5). Intracardiac digital exploration within the tremendous infundibular chamber revealed a normal pulmonary valve and artery, a very low infundibular obstruction, and a poststenotic dilated infundibular chamber the size of a lemon; the ventricular septum was intact. Digital dilatation with stellate splitting of the stenotic infundibular orifice was performed. Because of the low position of the infundibular obstruction, attempted resection was not successful.

The immediate postoperative condition was good and she was discharged within two weeks. In the fall of 1952 she developed subacute bacterial endocarditis which responded well to penicillin. Three and one-half years after surgery she had no complaints. Her appetite was normal and her tolerance to exercise had gradually increased to normal. The cardiac murmur and thrill were unchanged. Fluoroscopic examination revealed the presence of a small bulge in the infundibular region with no unusual pulsation.

CASE 2.—M. L., a 5-year-old white boy, was referred to St. Christopher's Hospital for Children on Oct. 19, 1952, for the surgical correction of pulmonic stenosis. A cardiac murmur had been present since birth. He had never been cyanotic. Dyspnea following exertion was noted at the age of 3 years.

Physical examination revealed a well-developed, fairly well-nourished child with a blood pressure of 100/80. A Grade 4, harsh systolic murmur was audible, loudest in the third left

TABLE III. ROENTGENOGRAPHIC OBSERVATION IN PULMONIC STENOSIS (INFUNDIBULAR)

	CARDIAC ENLARGEMENT	PULMONARY ARTERY SEGMENT	HILAR VESSELS	PERIPHERAL LUNG FIELDS	ANGIOCARDIOGRAM
Case 1, B. T., Female	None	Convex	Prominent pulmonary artery; quiet	Not Remarkable	Enlarged right ventricle and pulmonary artery; no shunts
Case 2, M. L., Male	None	Concave	Quiet	Clear	
Case 3, J. B., Female	Right atrium and ventricle	Concave		Clear	Prolonged retention of dye in right ventricle; no right to left shunt
Case 4, T. M., Male	None	Normal	Average; quiet	Not Remarkable	
Case 5, R. B., Female	None	Concave	Average; quiet	Clear	Selective; no injection of aorta; compatible with diagnosis of pulmonic stenosis with intact ventricular septum
Case 6, G. H., Female	Right atrium and ventricle	Concave	Right arch; average, quiet	Clear	Poor opacification of left cavities and aorta in dextroangiogram; late reopacification in levoangiogram delay dye in right ventricle; right arortic arch

interspace. A systolic thrill was palpable along the left sternal border. P_2 was diminished. (Table I.) There was no demonstrable cardiac enlargement.

The hemoglobin at the time of admission was 14.3 Gm., erythrocytes were 5.18 million per cubic millimeter, hematocrit 44. The circulation time was 9.8 seconds. Electrocardiographic tracings revealed peaked P waves in limb Lead II and evidence of right axis deviation and right ventricular hypertrophy.

Roentgenograms and fluoroscopic examination of the chest revealed the heart to be of normal size. The apex was upturned, the pulmonary conus was concave, and the hilar vessels were quiet. Vascular markings at the periphery of the lungs were reduced. The right ventricle was prominent as viewed in the left anterior oblique.

The results of cardiac catheterization (done elsewhere) are listed in Table II, and these suggested an infundibular stenosis with an intact ventricular septum.

Surgery was performed on Oct. 27, 1952. The pulmonary artery was the size of one's thumb, but there was no poststenotic dilatation. A thrill was palpable over the outflow tract of the right ventricle extending up into the pulmonary artery. Intracardiac exploration failed to reveal any valvular stenosis. There was an infundibular obstruction with an "infundibular chamber the size of a walnut" (Fig. 6). No other defects were noted. After infundibular resection (three bites) the pulmonary artery was more tense and the thrill over this area was harsher and more generalized.

The patient's immediate postoperative condition was good. Mild cardiac enlargement was noted roentgenographically. Seven months later he had steadily improved and his physical activity had increased considerably. Dyspnea following exertion was no longer a complaint. There were no significant changes noted over those found preoperatively in the physical examination, in roentgenograms, in fluoroscopic examination of the chest, or in the electrocardiographic tracings.

At present he is asymptomatic, although there is "some dyspnea on severe exertion." Repeat cardiac catheterization carried out fourteen months after surgery revealed the right ventricular pressures to have dropped from 75/0 to 40/0, infundibular chamber pressures from 40/0 to 30/0. The pressures in the main pulmonary artery were essentially unchanged; 26/15 before and 25/8 fourteen months later.*

*These catheterization data (pre- and postoperative) were obtained through the courtesy of Dr. Paul La Bissoniere, Marquette University School of Medicine, Milwaukee, Wis.

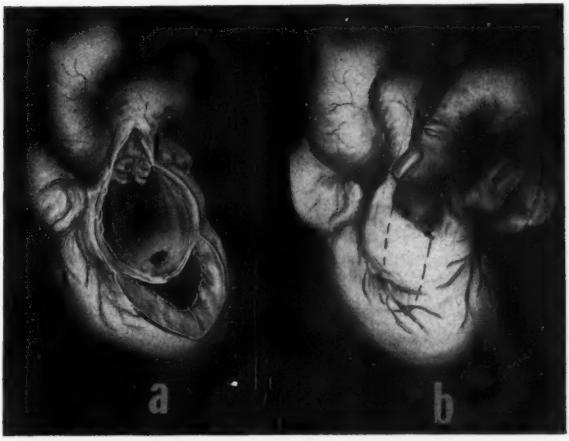


Fig. 5.—*a*, Unusually large-sized infundibular chamber (Case 1). In this instance the right ventricle was actually divided into two halves approximately equal in size. *b*, The stenosis could only be reached by the index finger which was thrust through the opening producing a stellate tear. No resection could be performed in this instance.

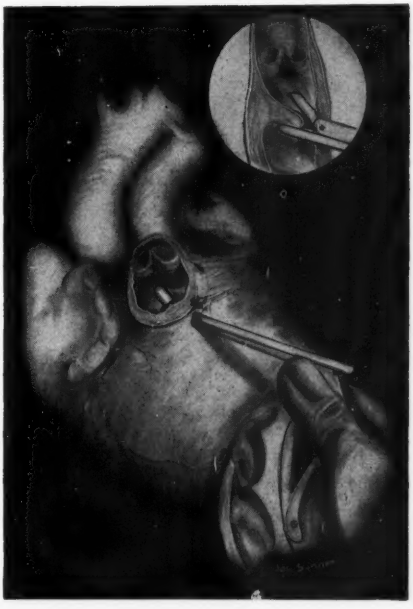


Fig. 6.—Typical walnut-sized infundibular chamber (Cases 2, 3, and 4).

CASE 3.—J. B., a 13-year-old white girl, had never been cyanotic, but a cardiac murmur had been noted since birth. At 10½ years of age she began "squatting" to relieve precordial pain and at 12 years exertional dyspnea and easy fatigue were noted.

Physical examination at the time of admission to Presbyterian Hospital revealed a well-developed, well-nourished girl with a blood pressure of 100/74. There was a harsh systolic murmur, loudest at the left base, and a systolic thrill was palpable along the left sternal border. The second sound in the pulmonic area was not split. (Table I.) Cardiac enlargement was noted on physical examination, as were congenital deformities of the hands and arms.

Her hemoglobin was 16 Gm.; erythrocytes were 5.27 millions per cubic millimeter. The electrocardiographic tracings revealed peaked P waves, right axis deviation, and evidence of right ventricular hypertrophy. Roentgenograms and fluoroscopic examination of the chest revealed cardiac enlargement, confined to the right atrium and ventricle. The pulmonary conus was concave and the vascular markings were reduced.

The results of cardiac catheterization and angiocardiography, done elsewhere, are listed in Tables II and III.

Surgery was performed on Jan. 22, 1953. The pulmonary artery was the size of one's index finger and very flabby. A rather faint thrill was palpable over the pulmonary artery, and proximal to the pulmonary valve there was an "egg-sized," thin-walled infundibular chamber where the thrill was maximal (Fig. 6). Intracardiac exploration revealed a normal pulmonary valve and a severe infundibular obstruction; the ventricular septum was intact. Extensive infundibular resection (four bites) was performed. The heart rate slowed, but picked up immediately after intracardiac manipulation ceased.

The immediate postoperative condition was good and two months later dyspnea had greatly reduced; the appetite had improved. The significant changes on physical examination were the absence of the thrill and the presence of a Grade 1 diastolic murmur over the pulmonic area. Roentgenograms revealed an obvious decrease in the heart size. Her hemoglobin was 13 Gm. and the erythrocytes 4.47 million per cubic millimeter. The electrocardiographic tracings revealed a migrating atrial pacemaker and a right bundle branch block in addition to the preoperative findings. These findings have now disappeared.

At present this patient is completely asymptomatic and lives a normally active life, including dancing at school parties. Recent cardiac catheterization carried out thirteen months after surgery revealed the right ventricular pressures to have dropped from 134/0 to 80/–1 in the mid-right ventricle and 80/0 high in the right ventricle. The pressure in the right pulmonary artery was 15/7.*

CASE 4.—T. M., a 6-year-old white boy, was admitted to St. Christopher's Hospital for Children for cardiac evaluation. A cardiac murmur was first noted at the age of 3 years. He had never been cyanotic. He had no cardiac symptoms.

Physical examination revealed a well-developed, well-nourished boy with a blood pressure of 92/60. A systolic thrill was palpable along the left sternal border from the second to the fourth interspace and a harsh Grade 4 systolic murmur was maximal at the third left interspace. (Table I.) P_2 was diminished and frequent premature contractions were heard.

The hemoglobin at the time of admission was 10.5 Gm., erythrocytes were 3.78 millions, the hematocrit 35. The electrocardiographic tracings revealed right axis deviation, right ventricular hypertrophy, and frequent premature contractions. Roentgenograms and fluoroscopic examination of the chest revealed the heart to be of normal size and contour; the hilar vessels were of normal caliber and quiet.

The results of cardiac catheterization are listed in Table II.

Surgery was performed on Sept. 28, 1953. At operation the pulmonary artery was of normal size; an infundibular obstruction could be demonstrated 3 cm. proximal to the pulmonary valve (Fig. 6). On palpation a rough systolic thrill was maximal over the outflow tract of the right ventricle propagated faintly to the pulmonary artery. Intracardiac ex-

*These catheterization data (pre- and postoperative) were obtained through the courtesy of Dr. Chester Kurtz at the Wisconsin General Hospital, Madison, Wis.

ploration revealed a diaphragm-like obstruction with a poststenotic infundibular chamber. The pulmonary valve appeared normal; no ventricular septal defect was noted. Following infundibular resection (four bites) the tension in the pulmonary artery increased.

The immediate postoperative condition was good, although the patient developed mild cardiac enlargement and early signs of decompensation, which immediately responded to digitalization. Four months later he was in full activity, without medication, and had no complaints. At four and at six months following surgery there were no changes on physical examination. Roentgenograms and fluoroscopic examination of the chest revealed mild cardiac enlargement, especially of the right atrium on frontal view; there was mild prominence of both ventricles in the oblique views. The hilar vessels were adequate and pulsating. The electrocardiographic tracings revealed no changes.

At present he remains asymptomatic and normally active as he was prior to surgery.

CASE 5.—R. B., a 6-year-old white girl, was admitted to St. Christopher's Hospital for Children because of cyanosis and dyspnea on exertion since the age of 4 years. She was not cyanotic at birth. A cardiac murmur and enlargement were first noted at 6 months. A few months prior to admission, clubbing of the fingers and toes was noted.

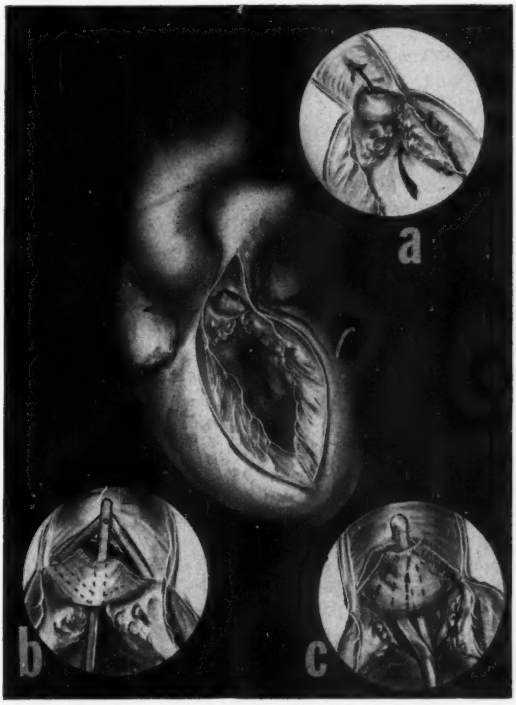


Fig. 7.—a, Infundibular (subvalvular) and valvular stenosis (Case 5). The infundibular obstruction was too close to the valve for safe resection, hence both stenoses were opened by the valvular technique (*b*) and then dilated to the maximum size of the artery (*c*).

Physical examination revealed a small, thin female with moderate cyanosis and early clubbing of the fingers and toes. A systolic thrill was palpable along the left sternal border; a Grade 3 harsh systolic murmur was maximal at the second and third left interspaces. (Table I.) P_2 was diminished; the blood pressure was 98/60.

Hemoglobin was 16.7 Gm., erythrocytes were 6.2 millions per cubic millimeter, the hematocrit was 53. The electrocardiographic tracings revealed peaked P waves in limb Lead II, right axis deviation, and evidence of right ventricular hypertrophy. Roentgenograms and fluoroscopic examinations of the chest revealed the heart to be of normal size; the conus was concave and the vascular markings reduced. In the oblique views there was marked prominence of the right ventricle.

The results of cardiac catheterization and selective angiocardiography are listed on Tables II and III.

Surgery was performed on Oct. 26, 1953. The pulmonary artery was the size of one's little finger and there was no poststenotic dilatation. The right and left branches were larger than the main pulmonary artery. The pulmonary artery was severely constricted as it emerged from the right ventricle, and a systolic thrill was palpable along the trunk. The usual cone-like pulmonary valve was not palpable. Intracardiac exploration revealed the obstruction to be immediately subvalvular; the ventricular septum was intact (Fig. 7). Valvotomy was performed through the infundibular and valvular obstructions followed by extensive dilatation to a size of 2 cm. by actual intraluminal measurement. The constricted region was now filled out; the pulmonary artery seemed tense and full and the thrill became more diffuse.

Postoperatively she developed early signs of decompensation and was digitalized. Two months later physical examination revealed decrease in the cyanosis and the disappearance of the thrill, otherwise the findings were the same as preoperatively. Fluoroscopic examination of the chest revealed adequate hilar vessels with normal pulsations. Digitalis therapy was discontinued and activity increased.

At present she is practically asymptomatic and acyanotic. Further time must elapse before her ultimate status can be ascertained.

CASE 6.—G. H., a 3-year-old Negro girl, was admitted to St. Christopher's Hospital for Children because of exertional dyspnea, easy fatigability, squatting, and questionable cyanosis on exertion. A cardiac murmur was first noted at the age of 2 years.

Physical examination on admission revealed a small, thin girl with a blood pressure of 86/60. A Grade 3 harsh systolic murmur was maximal at the second and third left interspaces; a systolic thrill was palpable in the same location. P_2 was clear. (Table I.)

Hemoglobin was 17.2 Gm., erythrocytes 5.1 millions per cubic millimeter, the hematocrit 55. The electrocardiographic tracings revealed right axis deviation and evidence of rather severe right ventricular hypertrophy. Roentgenograms and fluoroscopic examination of the chest revealed a mild cardiac enlargement with prominence of the right atrium and ventricle. The conus was slightly concave and the vascular markings were reduced. A right aortic arch was noted.

The results of cardiac catheterization and angiocardiograms are listed in Tables II and III.

Surgery was performed on Nov. 23, 1953. At operation the pulmonary artery was 12 mm. in diameter, very short, and had an area of constriction in its mid-portion. A rather faint thrill was palpable in the pulmonary artery, maximal immediately distal to the pulmonary valve. Intracardiac exploration revealed an infundibular obstruction with a poststenotic infundibular chamber the "size of a peanut" (Fig. 8). The pulmonary valve appeared constricted and the ventricular septum was intact. Valvotomy, infundibular resection, and dilatation of the constricted segment of the pulmonary artery were performed. The infundibular chamber and the pulmonary artery became more full and tense, the thrill increased in intensity and became more widespread.

Postoperatively she developed right-sided failure (palpable liver) and was placed on digitalis with good response. Two months later digitalis was discontinued and physical activity increased. Four months following surgery she was in full activity and had no complaint. Squatting was no longer noted. Physical examination revealed essentially the same findings as preoperatively except for increased intensity in the murmur and thrill. Roentgenograms and fluoroscopic examination of the chest revealed a mild cardiac enlargement and increased vascular markings.

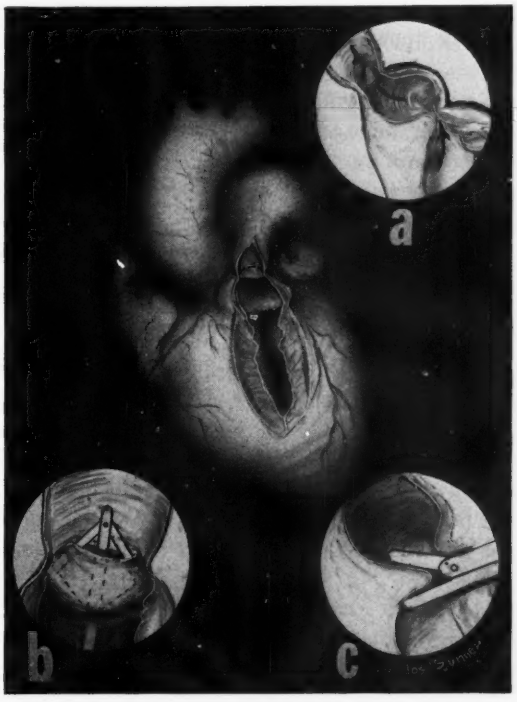


Fig 8.—*a*, Combined infundibular and valvular stenosis with an intervening "peanut-sized" chamber (Case 6); *b* and *c*, the valvular incision and the infundibular resection.

COMMENTS

Half of the patients gave a history of "squatting." Easy fatigability and dyspnea on exertion were the major complaints in all but one patient. There was no apparent relation between the height of the pressures in the right ventricle and the severity of the symptoms. This lack of correlation has been observed by others.[30] Pain in the precordium, cyanosis, and early clubbing were each noted on one occasion. There was moderate polycythemia in two patients and mild polycythemia in one.

Growth and nutritional status were mildly impaired in two patients. In all instances the systolic murmur was loudest either in the second or third left interspace and the thrill was palpable along the upper left sternal border. The second sound in the pulmonic area was pure and diminished in every instance.

Right ventricular hypertrophy was observed by roentgenographic means in all patients, but the cardiac area was increased in only two; these were those who presented evidence of right atrial enlargement. The conus area was concave in four, convex in one, and normal in the remaining patient. In only one instance was the pulmonary artery enlarged. The hilar vessels were

uniformly quiet. The lung fields showed decreased vascular markings in four instances; in two they appeared normal. A right aortic arch was present in one patient.

Right axis deviation and evidence of right ventricular hypertrophy were seen in all electrocardiographic tracings. Peaked P waves equal to or greater than 2.5 mm. were present in one-half of the tracings. Frequent premature contractions were seen in one patient; these persisted postoperatively.

In three cases conventional angiocardiography revealed prolonged retention of dye in the right ventricle. Early, rather poor opacification of the aorta was seen in one additional patient, the result of a shunt from the right to the left atrium; this was the only such shunt demonstrable by the dye studies.

The pressures in the right ventricle were moderately to markedly elevated in five of the patients. The pulmonary artery was entered in three patients. and in these the pressure was either normal or decreased. A gradual rise in the systolic pressure in the right ventricle was noted in three patients as the catheter was withdrawn. The systemic systolic pressure was near or under 100 mm. Hg in all patients and the pulse pressure was never greater than 30 mm. Hg. In three patients the systolic pressure in the right ventricle exceeded the systemic systolic pressures. No evidence of any left-to-right shunt was obtained by cardiac catheterization; a right-to-left shunt at the atrial level was suggestive in four patients (femoral artery oxygenation below 90 to 94 per cent) but questionable in two (Cases 2 and 4, Table II).

The diagnosis of pulmonic stenosis in this series was made on the basis of right ventricular hypertension and a diminished pressure in the pulmonary artery in those patients in whom the pulmonary artery was entered. In the remaining three patients the diagnosis was suggested by a combination of the clinical and laboratory findings, including right ventricular hypertension and roentgenographic evidence of a decreased pulmonary blood flow.

The presence of an intact ventricular septum was assumed from the oxygen studies which revealed an absence of any left-to-right shunting at the ventricular level. The finding of a systolic pressure in the right ventricle significantly higher than the systemic systolic pressure gave additional support to the presence of an intact ventricular septum in three cases.

The infundibular location of the stenosis was suggested by (1) the finding of a pressure gradient as the catheter was withdrawn from the pulmonary artery, and/or infundibulum, (2) difficulty in catheterizing the pulmonary artery, (3) concavity of the conus or absence of poststenotic dilatation, and (4) localized angiographic evidence of a small pulmonary artery with infundibular irregularities.

The diagnosis of an interatrial communication was proved or suggested (1) by the direct passage of the catheter from right to left atrium or (2) by the determination of the arterial oxygen saturation which revealed a peripheral arterial unsaturation in the presence of an intact ventricular septum.

At operation the ventricular septum was found intact by probing in all instances. In Cases 1 to 4 there was, in addition, considerable narrowing of the trunk of the pulmonary artery. Case 5 differed from the others in that the stenosis was high in the infundibulum (subvalvular) and thus presented a difficult surgical problem. In Case 6 the presence of a high systolic pressure in the infundibular chamber suggested that the significant obstruction was valvular or arterial in addition to a less significant infundibular stenosis.

RESULTS

The patients (Cases 1, 2, and 3) who have been followed for the longest period of time (1½ to 3½ years) have shown definite improvement, as indicated by an increase in exercise tolerance and a disappearance of symptoms. In one patient no functional change could be noted after seven months, as this patient had no initial cardiac disability. The murmur changed in intensity but remained present in all patients, and the systolic thrill disappeared in two patients. Mild cardiac enlargement developed postoperatively in four patients. In one in whom cardiac enlargement was present prior to surgery the size of the heart has subsequently become normal (Case 3); this was the only patient who postoperatively developed a diastolic murmur possibly indicative of pulmonary insufficiency. Following surgery she developed a transient migratory atrial pacemaker rhythm and a right bundle branch block. Cyanosis of diminished intensity persisted in one case (Case 5) and polycythemia disappeared in another (Case 3). The effects of surgery on polycythemia had not been determined in two patients (Cases 5 and 6). Three patients (Cases 4, 5, and 6) developed evidence of beginning right-sided cardiac failure which responded well to digitalis and rest; these measures were discontinued within a few weeks. Subacute bacterial endocarditis occurred in one patient seven months after surgery and was controlled by antibiotic therapy.

In summary, therefore, four patients are now functionally normal, and the two other more recent cases show definite improvement, as of seven and six months, respectively.

SUMMARY

1. The literature dealing with pulmonic stenosis has been reviewed and its incidence and pathophysiologic changes presented.

2. The diagnostic features of pure pulmonic stenosis are discussed with special emphasis on the differential diagnosis between this entity and the trilogy of Fallot.

3. Methods which aid in the determination of the site of the pulmonic stenosis are outlined.

4. The technique of right ventricular and pulmonary artery exploration with subsequent infundibular resection is detailed in the light of an increased experience with this type of vascular obstruction.

5. Twenty-five cases of pulmonic stenosis with intact ventricular septum have been treated surgically. In nineteen of these cases pure valvular steno-

sis was present; in four, pure infundibular stenosis was the obstructing lesion; and in two, both forms of stenosis were encountered. There was no mortality in the entire series.

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(Discussion follows the article by Swan, Cleveland, Mueller, and Blount: Pulmonic Valvular Stenosis, page 512.)

PULMONIC VALVULAR STENOSIS

Results and Technique of Open Valvuloplasty

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PULMONARY valvular stenosis with intact interventricular septum has been recognized with increasing frequency in recent years. Usually the patient is cyanotic, although occasionally a patent foramen ovale (or rarely a true atrial septal defect) may allow a right-to-left shunt of blood and thus peripheral arterial oxygen desaturation. All degrees of severity of the malformation apparently occur.[1] Many of the patients are asymptomatic for long periods of their lives, others die in infancy. In the more pronounced malformations, however, the great work-load imposed upon the right ventricle results first in its progressive hypertrophy, later in cardiac failure and death. The long-term importance of the lesion to the patient and the threat to his health is not necessarily reflected in the symptomatology, since decompensation may occur rapidly. The anomaly, therefore, when identified, cannot be considered benign. Complete physiologic evaluation is advisable and operative intervention considered in every case on the basis of such evaluation. We have recommended operation in all patients in whom the right ventricular systolic pressure exceeded 75 mm. Hg, and who showed a characteristic abrupt pressure gradient across the pulmonary valve, irrespective of the presence of symptoms.

Since the reports of Sellors[2] and Brock,[3] the transventricular blind instrumental manipulation of the congenitally deformed valve has become the commonly practiced method of surgical intervention. That this is an operation which can be done at low risk has been amply demonstrated.[4-6] The purpose of the operation is to relieve the stenosis of the pulmonary valve. How efficiently it does so has been a question on which evidence has ben slow in accumulating. Only within the past year have objective data been presented evaluating this procedure. In the light of these studies, it is now apparent that the transventricular instrumentation of the pulmonary valve in so-called "pure" valvular stenosis, as commonly performed in many cardiac centers in this country, produces quite variable results and fails in a high percentage of cases to relieve the valvular stenosis.

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It must be emphasized again that clinical evaluation of the effectiveness of this procedure is unreliable, just as it is unreliable in establishing the indications for the operation. The statement of the parents that the child is "better" or "more active" is notoriously colored by wishful thinking. Indeed, in many patients there were few if any symptoms present before operation; postoperatively, they are at best no worse. To determine this point requires evidence on the absolute and relative pressures existing in the right ventricle and pulmonary artery before and after valvulotomy. This evidence must be obtained before and some time after operation, *not at the time of surgery,* since data secured at the operating table after manipulation of the heart, ventriculotomy, and loss of blood may be misleading. This is well known to all who have taken pressure readings during the operation following extensive cardiac manipulation and compared these with subsequent observations by cardiac catheterization following convalescence.[7] Nothing short of preoperative and postoperative cardiac catheterizations, therefore, will yield reliable evidence on which to judge the degree of operative relief of pulmonary valvular stenosis.

About a year and a half ago, our own data obtained in this fashion began to give us cause for concern. At this time, we were performing the transventricular technique using the instrument devised by Potts.[8] In the light of this data, we were radical in our attitude in the manipulation of the valve, making cuts with the knife in at least two places, and attempting to further dilate the valve with vigor. In spite of this attitude, the results were neither uniform nor, in our opinion, adequate. For this reason, as soon as the method of operating during hypothermia with the cessation of circulation had reached the stage in our laboratory where we felt justified to give it clinical trial, this anomaly was one of the first to be attacked by direct vision intervention via an incision in the pulmonary artery. Since that time we have had the opportunity to operate on seven patients with "pure" valvular stenosis. There have been no deaths in this group. The first five patients have been thoroughly studied since surgery and provide a measure of the effectiveness of the procedure. On the basis of the comparison of the results obtained in these five patients with our own previous results and the published results of others, we feel at the present time that the transarterial direct vision approach is clearly the operation of choice.

EVALUATION OF TRANSVENTRICULAR VALVULOTOMY

In Table I we have recorded all of the data which we have found in the literature, together with data on five patients of our own in whom transventricular pulmonary valvulotomy has been performed for "pure" valvular disease, and in whom pre- and postoperative pressure studies of the right ventricle and pulmonary artery have been performed by catheterization.

In this table the "per cent reduction" of right ventricle pressure was computed as follows: We arbitrarily elected to choose 30 mm. Hg as the top of normal systolic pressure for this chamber. A perfect result would be lowering of systolic pressure to this level or lower. The degree to which the high

TABLE I. PHYSIOLOGIC OBSERVATIONS BEFORE AND AFTER TRANSVENTRICULAR PULMONARY VALVULOTOMY

AUTHOR	PREOPERATIVE		POSTOPERATIVE			
	R. V. PRESSURE	P. A. PRESSURE	R. V. PRESSURE	P. A. PRESSURE	PER CENT REDUCTION R. V. PRESSURE	RESIDUAL STENOSIS R. V.-P. A. (SYSTOLIC)
Lurie and Shumacker[9]	120/0	2/-2	101/0	3/-2	21.0	98
	65/-7	16/4	39/-5	18/6	74.0	21
	82/-6	46/5	44/-6	26/11	73.0	18
	105/4	46/13	106/0	33/12	-1.0	73
	55/0	19/3	48/6	20/9	28.0	28
	127/-7	5/-8	84/-3	14/4	44.0	70
	142/2	17/5	122/-7	14/5	18.0	108
Bing et al.[10]	165/36		135/39		22.0	
	122/30		100/3		24.0	
	84/60		37/14	20/13	87.0	17
	126/40		92/34		35.0	
	145/3	24/9	111/5	35/12	30.0	76
	118/13	22/9	90/10		32.0	
	166/26		72/8	20/9	70.0	52
	128/0	30/17	38/2	26/11	92.0	12
	90/0		98/12		-13.0	
Kirklin[7]	196/5	11/8	65/8	16/12	79.0	49
	55/5	18/10	40/5	20/12	60.0	20
Galligan et al.[11]	260/0		60/0		87.0	
	99/11		58/6		59.0	
Humphreys et al.[6]	217/9		69/2		79.0	
	162/12		78/12		64.0	
	201/16		77/13		73.0	
	100/0*		40/0*, 77/4		33.0	
Blount et al.[12]	200/5	16/20	151/1	22/13	29.0	129
	166/-7	22/13	38/2	25/10	94.0	13
	221/10	18/11	60/6	35/10	84.0	25
	142/-1	13/6	34/-5	14/8	96.0	20
	110/3	16/7	40/2	14/9	88.0	26

*Obtained at the operating table.

preoperative pressure was, in fact, lowered toward this level postoperatively was observed. The actual lowering compared to the desired lowering is expressed as "per cent reduction." The formula for "per cent reduction," therefore, is: Per cent reduction =

$$\frac{\text{Decrease in R. V. systolic pressure}}{\text{Preop. R. V. systolic pressure minus 30 mm. Hg.}} \times 100$$

The "residual stenosis" was computed in terms of the postoperative difference between right ventricular systolic pressure and the pulmonary artery systolic pressure. Presumably, if no stenosis at all is present, these two systolic pressures will be equal, as in the normal individual. Any gradient across the valve must represent stenosis, and the magnitude of the gradient may be considered to be a rough measure of the degree of the stenosis. It would be considered that less than 10 mm. Hg gradient would be insignificant, 10 to 40 mm. Hg would be moderate, while over 40 mm. Hg would represent significant residual stenosis.

It will be apparent, from a glance at Table I, first, that the results obtained are extremely variable; second, that almost one-half of the patients failed to

receive a 50 per cent reduction in their right ventricular hypertension, and only one-fourth of them received 80 per cent or better; and, third, that complete, or even near-complete elimination of pulmonary valvular stenosis is never accomplished.

We feel that this evidence strongly suggests that in the hands of at least several competent cardiac surgeons, transventricular valvulotomy fails to accomplish the desired result in many instances. Moreover, it is our feeling that, although the pulmonary stenosis may have been modified somewhat, patients who have residual right ventricular systolic pressures of 60 to 150 mm. Hg have not been adequately relieved of their disease and do not have a normal prognosis.

EVALUATION OF TRANSARTERIAL DIRECT VISION VALVULOPLASTY

In Table II, the data on our first five patients undergoing transarterial valvuloplasty under direct vision are presented. In these patients, body temperature was lowered to below 30° C.; the circulation entering the heart was temporarily occluded; through an incision in the main pulmonary artery, the valve was visualized; and a deliberate plastic reconstruction of the conical deformity was performed.

TABLE II. PHYSIOLOGIC OBSERVATIONS BEFORE AND AFTER TRANSARTERIAL PULMONARY VALVULOPLASTY

	PREOPERATIVE		POSTOPERATIVE			
PATIENT	R. V. PRESSURE	P. A. PRESSURE	R. V. PRESSURE	P. A. PRESSURE	PER CENT REDUCTION R. V. PRESSURE	RESIDUAL STENOSIS R. V.-P. A. (SYSTOLIC)
R. H.	93/3	23/12	29/0	23/8	102.0	6
J. K.	142/-2	17/7	19/-5	15/3	110.0	4
G. S.	110/3	18/12	32/-1	32/9	98.0	0
M. G.	119/-5	18/9	29/7	25/9	101.0	4
D. M.	115/54		29/7	15/9	101.0	14

In this table, the uniformity of the results, the completeness of the reduction in right ventricle pressure, and the almost complete elimination of gradient across the valve are apparent. Configuration of pulmonary artery pressures on catheterization shows a return to normal contour (Fig. 1). We feel that these data are strong evidence of the success achieved by this method in the relief of pure congenital pulmonary valvular stenosis.

TECHNIQUE

The methods for control of the hypothermic patient and the protective measures taken to prevent ventricular fibrillation and coronary air embolism have been previously described.[13-16] The transverse sternal-splitting incision through the fourth intercostal space on the right and the third intercostal space on the left has proved to give the best exposure in most instances. After the exposure and pericardiotomy are completed, the deformity confirmed by inspection and palpation, the azygos vein ligated, and tapes placed around the two cavae, the curved toothed aortic clamp of Potts is placed on a bite of main pulmonary artery in a vertical direction just distal to the valve ring. This is facilitated by first cleaning off the adventitia in the area by sharp dissection and then

placing four control sutures of 5-0 silk in the wall of the artery, one at each end and one in the middle on each side of the proposed 2 cm. incision. After the clamp has been placed, the incision into the artery can be completed before circulation is occluded. When all is in readiness, the tapes on the venae cavae are tensed, cutting off the inflow of blood to the heart. After thirty seconds, a noncrushing clamp is placed across the aorta about 2 cm. distal to its origin, and 0.5 ml. of 1:4,000 Prostigmin is then injected into the aorta between the valve and the clamp, taking fifteen to twenty seconds for the injection. This aortic clamp is now removed. A straight-ended auricular clamp of Harkens is now applied to the very base of the aorta, attempting to occlude the coronary

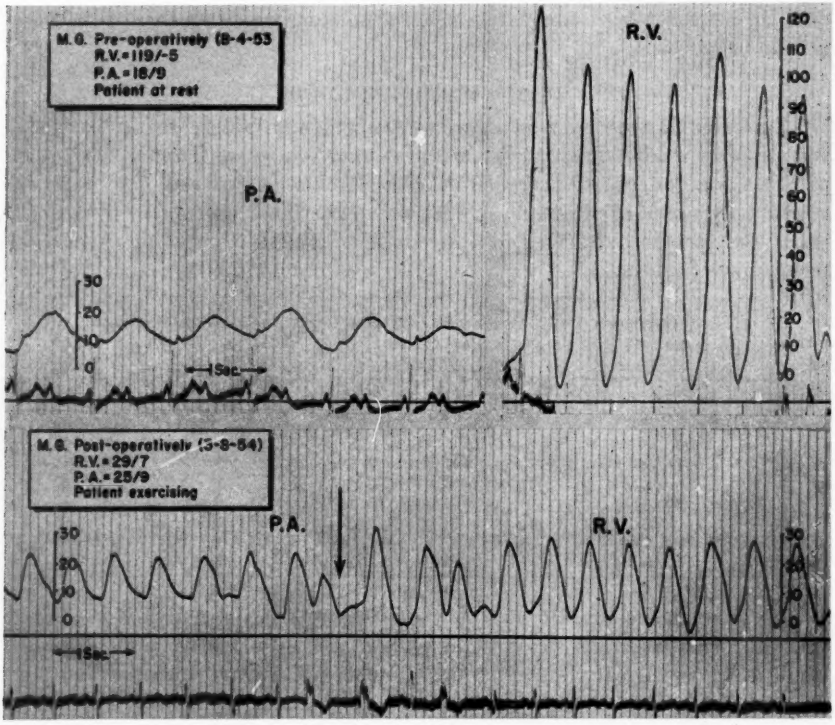

Fig. 1.—Pre- and postoperative recordings of right ventricular and pulmonary artery pressure tracings in patient M. G. Seven months following direct vision pulmonary valvuloplasty, pulmonary hemodynamics are essentially normal, even during exercise.

ostia in its grip. An angle-toothed ductus or aortic clamp of Potts is now applied to the pulmonary artery distal to the incision in that vessel. The originally placed curved clamp protecting the incision is now removed, allowing entrance into the lumen of the pulmonary artery at approximately one minute after onset of cessation of circulation.

The heart is beating slowly, perhaps 4 to 10 times a minute. The first two or three contractions will serve to empty the right ventricle of blood; from that point on, the operative field is essentially dry. The typical conical valvular deformity lies in clear view. With the tip grasped with toothed forceps, an incision is made with the scissors from the lumen of the valve all the way to the valve ring. If the rim of the lumen is thickened, or if the cone is unduly long, the distal segment of the valve is excised. A

second cut 180° from the first is now made, again making sure that the incision extends completely to the ring. The valve now consists of two cusps with complete commissures. A finger or an instrument can and should now be inserted through the valve into the right ventricle to ensure the absence of concomitant infundibular stenosis. This entire maneuver, since it is usually relatively simple, can be performed in from one to three minutes, without haste.

Satisfied that the plastic revision of the valve is adequate, withdrawal is accomplished by the following steps. Traction upward is exerted on the four control sutures in the pulmonary artery. The entire chest cavity is flooded with Ringer's solution poured from a large pitcher, making certain that the heart is filled and that all air is pumped out through the open pulmonary artery, which is about the uppermost portion of the heart when the patient is lying on his back. The curved-toothed clamp is reapplied to the incision, if possible under the surface of the Ringer's solution, taking care not to take so big a bite that obstruction to blood flow will occur. The control instruments are released in order: first, the tape on the superior vena cava (to allow blood to mix with the Ringer's solution), then the clamp across the base of the aorta, then the clamp across the pulmonary artery, and, finally, after an additional minute or two, the tape on the inferior vena cava. Blood flow is now fully restored.

This entire procedure can usually be performed during three to four minutes of total circulatory occlusion, together with an additional minute of inferior vena caval occlusion. After the first few beats, the heart rate will usually begin to increase as the myocardium becomes more pink, and the Prostigmin effect gradually wears away.

The incision in the pulmonary artery may now be closed with interrupted sutures of arterial silk. After hemostasis is adequate, routine closure of the thorax follows insertion of a penicillin-streptomycin solution and the placement of bilateral Pezzar catheters for postoperative pleural drainage through stab wounds. We tend to put no dressing over the incision. If warming of the patient in the tub of warm water is contemplated, collodion is used to seal the incision, and the finger of a rubber glove is sealed to the chest wall around the chest tubes with dermatome glue to avoid the possibility of the patient sucking unsterile water into the pleural space around these catheters.

Postoperative care is similar to that of any clean transthoracic procedure.

POSTOPERATIVE CLINICAL OBSERVATIONS

After convalescence, examination of the patients revealed some interesting changes. The thrill present preoperatively in the second left intercostal space disappeared entirely, and the harsh systolic murmur decreased markedly in intensity. The second heart sound, previously decreased or absent, was now of normal intensity. A diastolic murmur, heard best in the third left intercostal space, low in pitch and rough in quality, about Grade 2 in intensity, could be heard in four of the five patients.

Laboratory studies included electrocardiograms and x-rays. The former showed evidence of decreased right ventricular hypertrophy, and in three patients a configuration suggesting incomplete right bundle branch block was noted. The chest films revealed an increase in the size of the heart in all patients, ranging from 0.5 to 1.5 cm. in the transverse diameter. This increase occurred immediately after surgery and did not progress in the months that followed. It was of the same order of magnitude that we had previously observed following the transventricular procedures. It was observed whether or not a diastolic murmur in the left third intercostal space was heard.

All the children were asymptomatic, leading the vigorous life of childhood without restraint.

DISCUSSION

Isolated pulmonary valvular stenosis is one disease in which precise physiologic data are essential in order to establish properly both the indications for surgery and the results obtained by operation. Objective data secured at operation are unreliable. (See Table I, Humphreys and associates,[6] Case 4, with an operative finding of right ventricular pressure postvalvulotomy of 40/0, whereas later, by catheterization, it was seen actually to be 77/4.) Pre- and postoperative catheterization appears to be essential. On the basis of data obtained in this fashion, transarterial valvuloplasty would seem to achieve complete relief of pulmonary valvular stenosis.

The theory expressed by Kirklin and his colleagues[7] that muscular hypertrophy of the right ventricle may be so great as to cause obstruction to the outflow tract, thus explaining residual postvalvulotomy right ventricular hypertension, is not substantiated by our findings. Although this may occasionally occur, it appears that if the operation on the valve is adequate, the pressure in the right ventricle will promptly fall to normal, as occurred in our five patients. Bing and his co-workers[10] also cast doubt on this theory, since their molds of the lumina of the heart chambers in pure pulmonary valvular stenosis showed no infundibular obstruction due to hypertrophy. The simultaneous occurrence of congenital obstruction in both sites does, of course, occur in rare instances.

The appearance of a diastolic murmur in the pulmonic area in four of these children, we believe, is strong suggestive evidence of the creation of some degree of organic pulmonary valvular insufficiency. In the first three patients operated upon with this method, we made no effort to prevent this eventuality, since on theoretical and experimental considerations we believed that insufficiency of the pulmonary valve (unlike that of all other valves) would be of inconsequential significance. The regurgitant pressure in this instance is that of the diastolic gradient across the valve. In the human being, this might be in the range of 5 to 15 mm. Hg, a low pressure indeed. Over a period of several years we had attempted on occasion to create experimental heart failure by radical excision of the pulmonary valve in dogs. Although we kept some of these animals for years, no evidence of cardiovascular disturbance appeared. A similar experience was reported to us by Hufnagel.[17] For this reason, in our desire to effectively relieve stenosis, we excised considerable portions of the deformed valve in the first few patients. As we have increased our experience, however, we feel that it is probably possible in many instances to avoid insufficiency by attempting to shape a bicuspid valve which will close adequately. The two cuts completely through the valve, together with moderate trimming of the edge, may accomplish this plastic result. In our last three patients, the diastolic murmur has been absent or of low intensity.

In the evaluation of all of these patients, the benign nature of pulmonary regurgitation appears to be confirmed. The evidence is as follows: There appears to be no difference in the changes which occur in heart size following

the operation, whether or not the diastolic murmur is present. The heart diameter by x-ray moderately increases and then stabilizes. Right ventricular hypertrophy decreases as suggested by electrocardiographic evidence. This change occurs following the transventricular repair as well. The greater size probably indicates ventricular readjustment to increased flow. Second, the intensity of the diastolic murmur decreases with time. If intensity parallels insufficiency, readjustments apparently occur which diminish the degree of insufficiency. Third, as will be noted in Table II, the pulmonary valve is still capable of maintaining a diastolic pressure in the pulmonary artery 8 to 9 mm. Hg higher than that in the ventricle. These diastolic pressures are essentially normal. Certainly regurgitation is not complete, nor apparently even of significant degree.

On the basis of these observations, it is our opinion that the relief of stenosis is the primary objective of the operation and should be achieved even at the expense of slight or moderate insufficiency. Insufficiency should not be deliberately disregarded in the plastic repair, but elimination of stenosis by complete incision across the funnel-shaped valve is a sine qua non of the procedure.

SUMMARY

1. Seven patients with "pure" pulmonary valvular stenosis with intact ventricular septum have been subjected to pulmonary valvuloplasty under direct vision during hypothermia. All these patients have recovered from the operation.

2. The first five patients have had careful pre- and postoperative physiologic studies. The operation uniformly restores essentially normal hemodynamics.

3. Comparison of these observations with similar studies by ourselves and others of the results of transventricular valvulotomy apparently indicates superiority of the open technique for relief of this congenital valvular anomaly.

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Discussion

(Papers by Glover, O'Neill, Gontigo, McAuliffe, and Wells; and Swan, Cleveland, Mueller, and Blount)

DR. AARON HIMMELSTEIN, New York, N. Y.—I should like to discuss this as to the question of the adequacy of the Brock procedure, and would like to show three slides of tracings made in the operating room. This 7-year-old child was operated upon at the Mt. Sinai Hospital, New York.

(Slide) These tracings were taken by using a Brock probe that we had made in a hollow form and attached by lead tubing to a recording device. Here is a tracing taken before any manipulation of the valve had been done and you can see that the pressure in the pulmonary artery is 10/0; and the rapid transition to right ventricular pressure tracing which is recorded at 135/15. The second lead in the electrocardiogram is here.

(Slide) The valve was divided by using the Potts type knife of smaller size; it was passed into the pulmonary artery opened to its largest size, and the valve then was dilated, using the basket dilator, and this recording was made using the same Brock hollow probe. Here we see that the pulmonary artery pressure is 25/12; again there is still evidence of stenosis because the right ventricular pressure is 58/16. By our clinical criteria we felt we had done a very adequate operation, and yet here in the operating room we have distinct evidence of the fact that stenosis still is present. Therefore the larger sized Potts knife was introduced.

(Slide) This tracing was taken after the second size was opened to go through the valve at 18 mm. diameter, and here we see that we have lost the evidence of stenosis in this tracing taken by the same technique; the systolic pressure in the right ventricle is now 56 mm. Hg, and the peaks in the pulmonary artery are recorded at 53 mm. Hg.

Our point is that in the first pass we thought we had performed a very adequate operation, and in the second pass we proved we had to do a more adequate operation. We have no question in our minds that open procedure is preferable to any type of closed procedure if it can be done with the same safety, the same morbidity and mortality as the closed procedure.

DR. JULIAN JOHNSON, Philadelphia, Pa.—We, too, have been fairly well satisfied with our patients from a clinical standpoint but have been dissatisfied from the standpoint of the pressure readings which have been obtained. Our best results have been in infants or children where the Potts knife is sufficiently large to get a good sized opening. Although we have used a 22 mm. knife in some adults, we felt that we had not obtained an adequate opening according to our pressure readings.

(Slide) The Potts knife and dilator were not large enough in one particular patient, who was in his 30's, so we used the large Bailey aortic dilator and opened it in the pulmonary valve. Even under those circumstances and in spite of the fact that we put an index finger down in the ventricle to be sure that we did not have an infundibular

stenosis, and even though we could feel the stenotic valve, it was not opened adequately and the pressure was reduced less than 50 per cent. This indicated to us that the valve had an elastic quality and was pushed aside by the knife which perhaps was not sharp enough, and was dilated by the large aortic dilator, only to come back close to its original size.

(Slide) I present an experience with another technique even though it has been used on only one patient to date because we got phenomenal changes in the murmur and the pressures in the ventricle were reduced to 30 immediately postoperatively, and the pressure in the pulmonary artery raised to 25 mm. Hg. I believe one has a better opportunity of cutting the valve entirely back to the wall by using the small guillotine type Bailey mitral knife.

The knives come in pairs. The right-handed knife may be easily inserted through the ventricle and the valve engaged by the hooked blade of the guillotine. By pulling back slightly, the hooked end of the knife may be seated at the base of the valve at its junction with the vessel wall. The guillotine may then be closed, cutting the valve back to its base. The other side of the valve may be cut in the same manner with the left-handed knife.

While I have used it only one time to date, it seems like such a logical method and seemed to work so well that I thought it might be worth while to bring this experience with the method to your attention.

DR. CHARLES P. BAILEY, Philadelphia, Pa.—I had not intended to discuss this paper, but since Dr. Johnson has again proved that great minds tend to run on the same track, I thought I had better present the work of Dr. Henry Nichols, my associate. He, too, some months ago, thought of the same thing Dr. Johnson did, and applied the regular mitral commissurotomy guillotines in an adult with pulmonic stenosis of this type, and did obtain an excellent result so far as pressure reduction was concerned. This work will be presented at the meeting of the American College of Chest Physicians next month.

Dr. Nichols is not here and I do not have any slides showing this procedure. In small children one cannot insert a finger into the heart, so he combined the two sides of the guillotine to make it a double-cutting instrument. The wedgelike point goes up into the pulmonary artery, above the valve, then it opens on both sides. As it comes back it engages both leaflets and shears them off level with the pulmonary arterial wall. I think the secret of all this discussion is just what Dr. Johnson has said; if you cut the valve fully to the wall by a closed technique you get just as good a physiologic result as when you do it by a wide open technique with the aid of hypothermia or the heart-lung machine. I think all these presentations and comments have been most valuable in pointing out the inadequacies of the methods that have been used in the past.

DR. WILLIS J. POTTS, Chicago, Ill.—I am sorry Dr. Swan did not mention the ages of his patients. I am quite sure that the simple, old-fashioned transventricular valvulotomy is very effective in infants and young children because the stenotic valve is thin and easily cut or dilated to its base. We have operated upon fifty-two patients with "pure" pulmonary stenosis, with one death. Thirty were 4 years old or less; in fact, eleven children were less than 1 year of age at the time of operation, the youngest, 23 days. The valve was easily cut and dilated in these little children, often with no feeling of resistance to the instrument.

In older patients, especially those in the upper teens and older, the stenotic valve is fibrotic and tough and it may be difficult to open it all the way to its attachment on the wall of the pulmonary artery.

Thus far we have used the valvulotome and dilator introduced through the right ventricle in all but three cases, in which the instruments were introduced through the left pulmonary artery.

One cannot help but be concerned about the extent to which the valve is opened during a blind procedure. Time only will tell whether the rather formidable procedure advised and beautifully executed by Dr. Swan and his associates is justified on the basis of many factors which cannot be discussed now.

The children we have operated upon run and play, ride bicycles, fight, get into trouble, and seem to live normal lives. I guess we have made a bad mistake in not catheterizing them.

DR. EDWARD E. AVERY, Chicago, Ill.—With Dr. Swan's technique of resection of a portion of the pulmonary valve for pulmonary stenosis, we have been concerned about the production of pulmonary insufficiency and its effects. All the cases of pulmonary valvular insufficiency that I have been able to find in the literature have been from postmortem findings. Because of this, I would like to report one case discovered by cardiac catheterization. This 24-year-old medical student came in for study because of a loud diastolic pulmonic murmur. Catheterization revealed complete pulmonary valvular insufficiency with pressure of about 24/1 in both the pulmonary artery and right ventricle. He has been entirely asymptomatic, has had an athletic life, and his electrocardiographic findings are entirely normal.

Because of the excellent results that Dr. Swan has had with valvular resection, and because we were not set up for hypothermia, we thought that opening the valve in more than one plane might relieve ventricular hypertension even though it might produce some regurgitation. In one 31-year-old woman we opened the valve with the 22 mm. Potts valvulotome to 19 mm. in the usual horizontal plane, and then rotated the knife 90° and incised the valve to 19 mm. In spite of this two-plane incision opening of the valve, her catheterization findings three and one-half months postoperatively show her right ventricular pressure to be about 100/10, which is approximately the same as it was preoperatively. Clinically, however, she has had an excellent result.

I would like to ask Dr. Swan to comment on the amount of valvular tissue removed, whether these patients have a diastolic murmur postoperatively, and if he has any other remarks regarding long-term effects of pulmonary valvular insufficiency.

DR. HENRY SWAN, II, Denver, Colo.—I should like to thank the discussants for their remarks. With regard to the pressure taken at the time of operation, I think the pressures taken in the ventricle and pulmonary artery before any manipulative procedures on the heart are probably reasonably accurate. However, after any extensive manipulation has been done, including ventriculotomy, the pressures obtained are often quite unreliable and I think anyone who has taken pressures at the operating table and subsequently compared the pressures with catheterization results has been dismayed at the disparity between the two figures. Therefore I do not believe that anything short of careful study at some time postoperatively, when the circulatory dynamics are essentially back to normal, will give the actual situation.

I quite agree with Dr. Johnson and Dr. Bailey that it is highly desirable to cut the valve all the way to the ring. It is satisfying to be able to see and to know that you have done so.

Dr. Avery brings up a most important point that has been worrying us. In the first two or three patients I think we may have been enthusiastic in taking out pieces of these valves. I am not sure that we ever succeed in achieving a completely competent valve if it is cut all the way to the ring. Four of these five patients have Grade 2 diastolic murmurs along the left border of the sternum which were not there before operation.

(Slide) This gives some information about the degree of insufficiency in these patients. We have dogs now going into their fourth year with the pulmonary valve totally excised. I wish to point out that the pulmonary valve is able to hold up a good diastolic pressure in the pulmonary artery. The difference between the right ventricle

and the pulmonary artery diastolic pressure in these five patients was 8, 8, 10, here 2 and here 2. So in most of the patients the diastolic pressure in the pulmonary artery appears to be holding up.

The clinical observation is that, in the electrocardiogram, ventricular strain pattern essentially disappears. The heart, as shown by x-ray, enlarges somewhat within the first two weeks, exactly as seen following the Brock procedure; it then stabilizes and we think this represents its adjustment to increased flow. As you once so beautifully stated in a paper, Dr. Holman, "the heart and great vessels place themselves in harmony with the blood flow through them." And here we think we have considerably increased the blood flow so the right ventricle actually becomes larger so far as the lumen is concerned although its wall decreases in thickness. We have no experience in the long-term follow-up of pulmonary insufficiency, since this is a disease so rare in man.

Dr. Potts, I certainly agree that the clinical results in these patients are very good and you certainly will have helped almost all those children; I hope you will not have occasion to reoperate on too many of them in the years to come.

If you read back far enough you will find that Dr. Potts, like Dr. Rienhoff, has done almost every operation; and, not only that, he has designed a good instrument with which to do it.

EXPERIENCES IN THE SURGICAL TREATMENT OF AORTIC STENOSIS

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THE importance of developing a method for surgically treating aortic stenosis cannot be overemphasized, because acquired aortic stenosis, resulting from rheumatic heart disease, is the second most common acquired stenotic valvular deformity. Success in the surgical relief of other valvular heart lesions, particularly mitral stenosis, stimulated efforts to devise a method for the surgical treatment of aortic stenosis. Tuffier[1] is credited with the first clinical attempt to relieve aortic stenosis in 1913, but little consideration was given to the investigation of methods for relieving this disorder until the work of Smithy and associates[2] again focused attention on this problem. Smithy investigated the possibility of performing aortic valvulotomy in dogs through a small incision in the aorta immediately above the aortic valve, by means of a sheathed hook knife inserted through the valve cusp. He had no difficulty in incising the valve leaflet, but did have trouble controlling hemorrhage from the friable aorta of the dog because the sutures would tear out. Bailey and associates[3] approached the problem in various ways. They bypassed the valve in the experimental animal by means of an aortic graft from the left ventricle to aorta. However, these grafts thrombosed in the majority of animals. Using a cutting punch, they also approached the valve retrogradely by means of the brachiocephalic trunk and through an incision in the left ventricle, but uncontrollable regurgitation produced a large number of deaths.

One patient whose valve was incised died immediately of severe aortic regurgitation. They concluded that mechanical dilatation, rather than cutting the valve, seemed the most favorable method of surgically treating this disease. Although, initially, retrograde dilatation through the carotid artery was thought to be a satisfactory approach, difficulty was encountered later in finding the stenotic orifice of the inverted cone-shaped valve through this

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blind approach, and the investigators began to employ a transventricular approach through an incision in the myocardium of the left ventricle. Brock[4] also attempted to approach the valve retrogradely by passing a cardioscope into the carotid artery through an incision in the neck, with the idea of first visualizing the stenotic orifice and then incising the valve to relieve the stenosis. However, although he could visualize the valve leaflets and the surrounding aorta, he was unable to see the orifice with this instrument. Daniel and Scott[5] initially attempted to perform a digital commissurotomy of the aortic valve through an incision in the wall of the left ventricle, but the associated hemorrhage and the friable character of the left ventricular myocardium made this method too hazardous. They subsequently used a dilating instrument which they passed through a small incision into the aortic valve. Bailey and associates[6, 7] developed an instrument with parallel dilating bars which, in order to help locate the stenotic orifice, employed a lead wire that was first inserted into the ventricle and through the valve orifice so that the dilator would follow. More recently[8] they advocated a retrograde approach to the valve through a sternum-splitting or right thoracotomy incision, by means of suturing a plastic pouch to the wall of the aorta, through which the finger can be inserted simultaneously with the dilating instrument to palpate the valve and to guide the instrument into the stenotic orifice. They believe that this approach is the most desirable one in cases of isolated aortic stenosis.

During the past eighteen months we have operated on a series of patients with aortic stenosis, using a ventricular approach and employing a dilating instrument to open the valve. The results in this group of patients are presented.

PATHOLOGIC AND PHYSIOLOGIC CONSIDERATIONS

Aortic stenosis may be either acquired or congenital. The acquired type is usually the result of rheumatic heart disease and is seen much more frequently than the congenital type. Fusion of the valve leaflets occurs as the result of the rheumatic process,[9] and scarring, along with relatively early and intense calcification, often tends to distort the valve until residua of commissural fusion lines are difficult to demonstrate. Congenital aortic stenosis may occur as either valvular or subvalvular stenosis. The valvular type may be diaphragmatic in character, similar to the congenitally stenotic pulmonic valve, or occasionally may be a stenotic bicuspid valve, which is not infrequently seen with coarctation of the aorta. Subaortic stenosis is composed of a diaphragm of fibromuscular tissue, located immediately beneath the valve, which itself may often be normal.

In aortic stenosis the primary physiologic derangement is a marked increase in resistance to emptying of the left ventricle. This results in hypertension within the left ventricular cavity, which is often well tolerated for many years. There is apt to be a low aortic systolic pressure and a relatively normal diastolic pressure, resulting in a small pulse pressure. Reduced coronary blood flow accentuates the already tremendous strain imposed on

the left ventricle by the stenotic valve. If the mitral valve is also affected by the rheumatic process, mitral insufficiency is more likely to occur because of the increased left ventricular pressure. Over a period of time, left ventricular hypertrophy develops, placing increased demands on the already inadequate coronary blood supply.

SELECTION OF CANDIDATES FOR AORTIC VALVULOTOMY

A classification of patients with aortic stenosis, primarily according to the severity of their symptoms, may be of aid in selecting candidates to undergo aortic valvulotomy.

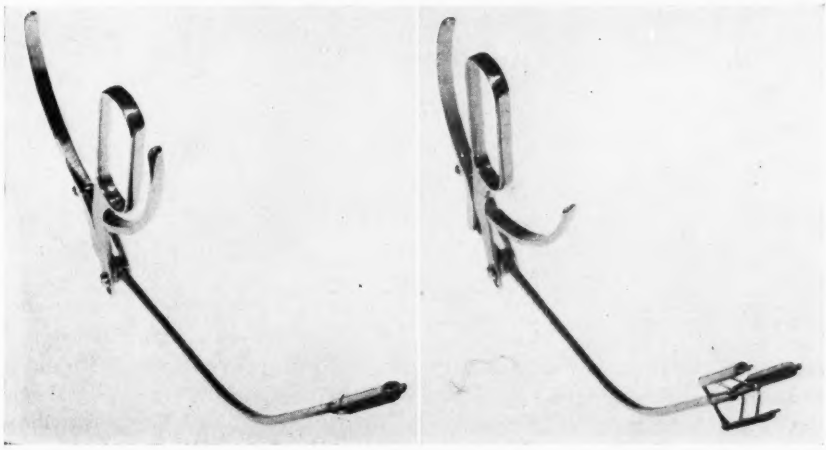
Group I. Patients with a murmur characteristic of aortic stenosis only.
Group II. Patients with a murmur, palpitation, easy fatigability, and mild dyspnea.
Group III. Patients with the more advanced symptoms of dizziness, angina, and syncope.
Group IV. Those with any of the above symptoms associated with an additional defect, such as mitral stenosis.
Group V. Those with advanced disease who have congestive failure and peripheral edema, in addition to the symptoms in the other groups.

Patients selected as candidates to undergo aortic valvulotomy by present techniques should have relatively pure aortic stenosis. If, in addition, a mitral valvular lesion is present, it should be predominately stenotic, although a considerable degree of insufficiency may be present. This insufficiency may be greatly decreased if the aortic valve can be opened satisfactorily. Many patients with aortic stenosis have diastolic murmurs characteristic of aortic insufficiency. However, unless other findings, such as a low diastolic pressure, an excessively enlarged left ventricle, and a great intensity of the diastolic aortic murmur itself, are present, the operation should be performed. Age, per se, is not necessarily a limiting factor. However, patients over the age of 50 or 55 years should be considered very cautiously. Patients with congenital aortic or subaortic stenosis should be operated upon when the diagnosis is made, if electrocardiographic and roentgenographic examination give evidence of left ventricular strain and enlargment. At present, we believe that patients who fall under Groups II, III, IV, and V should be considered as candidates for aortic valvulotomy. The regimen for those with severe symptoms and evidence of heart failure should consist of digitalis, mercurial diuretics, and bed rest before the operation in order to remove as much excess body fluid as possible. The heart size itself will often become much smaller and the lung fields less congested as a result of this treatment. It is important to operate on patients with aortic stenosis as early as possible, especially if their symptoms have been severe because, unlike patients with mitral stenosis, those with aortic stenosis, after only one or two episodes of failure, may develop fatal left ventricular failure. One patient who appeared to be in satisfactory condition for operation suddenly died of acute left heart failure the day prior to operation. He had just eaten and had felt well until this sudden failure

occurred. There are several contraindications to operation, such as marked aortic insufficiency, complete heart block, subacute bacterial endocarditis, evidence of very low myocardial reserve, acute rheumatic fever, and several others, depending upon the individual patient.

TECHNICAL CONSIDERATIONS

In the patients herein reported, the valve was dilated by an instrument (Fig. 1) inserted through a stab wound in the left ventricle. This approach was chosen because of the simplicity of the operative exposure and ease with which the valve can be entered from the ventricular side. The patient is placed supine on the operating table, and the left side is elevated approximately 20 degrees. Endotracheal anesthesia is used to maintain the patient after induction with cyclopropane or Pentothal sodium. The chest is entered through an anterolateral incision in the fifth intercostal space, extending from the left border of the sternum to the posterior axillary line. The fifth costal cartilage is incised adjacent to the sternum. The pericardium is incised, be-



A. *B.*

Fig. 1.—*A*, Closed aortic valvulotome. *B*, Aortic valvulotome with dilating components in expanded position.

ginning at the apex and extending superiorly to the origin of the aorta (Fig. 2). An avascular area in the myocardium of the left ventricle near the apex is infiltrated with 1 per cent Novocain. A mattress suture of No. 2 braided silk is placed in this area, so that a square of approximately 1.5 to 2 cm. of tissue is included. Deep bites into the myocardium are taken so that the suture will not pull through. Pressures are then recorded from the left auricle, left ventricle, and aorta, and a small stab wound is made in the center of the tissue within the mattress suture. The valvulotome is inserted through the stab wound, along the ventricular septum and into the aortic

valve (Fig. 3). One can easily feel the instrument after it passes into the aorta beyond the stenotic valve, which is usually located easily with the leading end of the valvulotome. However, only mild to moderate force should be employed to pass the instrument through the valve opening. With dilatation of the valve, one usually feels the sensation of sudden "giving" as it opens (Fig. 4). The heart may become momentarily slow, or occasionally more rapid. The instrument is withdrawn and the pressures are again

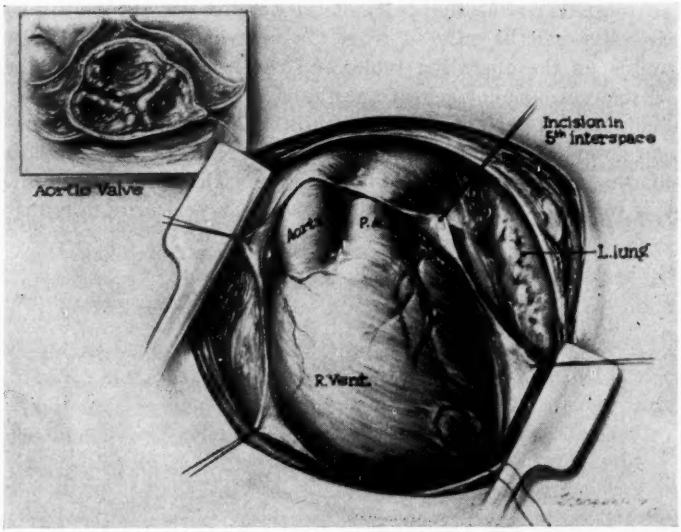
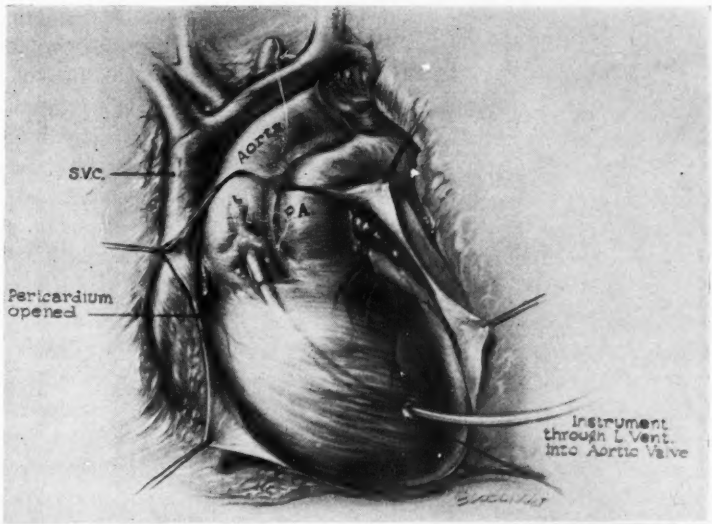


Fig. 2.—Drawing of operative exposure through anterior left fifth intercostal space. The pericardium has been incised from the apex to the origin of the great vessels. A purse-string suture has been placed in the left ventricular muscle.

measured. If there is a satisfactory change in pressures, the stab wound is closed with interrupted silk sutures and the purse string is removed. If not, the valvulotome is again introduced for further dilatation. The pericardium is closed loosely with interrupted sutures. A small window of pericardium is removed in the posterior inferior aspect to allow dependent drainage. Penicillin is placed in the chest cavity and the chest is closed in anatomic layers. An intrapleural catheter is attached to water-seal drainage.

This approach has the disadvantage of producing a wound in the ventricular myocardium, thus increasing the likelihood of serious arrhythmias, such as ventricular fibrillation. In this series we have not encountered this difficulty. Another disadvantage is that bleeding from the hypertensive left ventricle may be difficult to control. The purse string tore out on three occasions during early operations in this series, so we decided to attempt a retrograde approach through the innominate or carotid artery. This approach should have the advantages of better control of bleeding and not injuring the wall of the left ventricle. We were aware of the difficulties which both

Fig. 3.



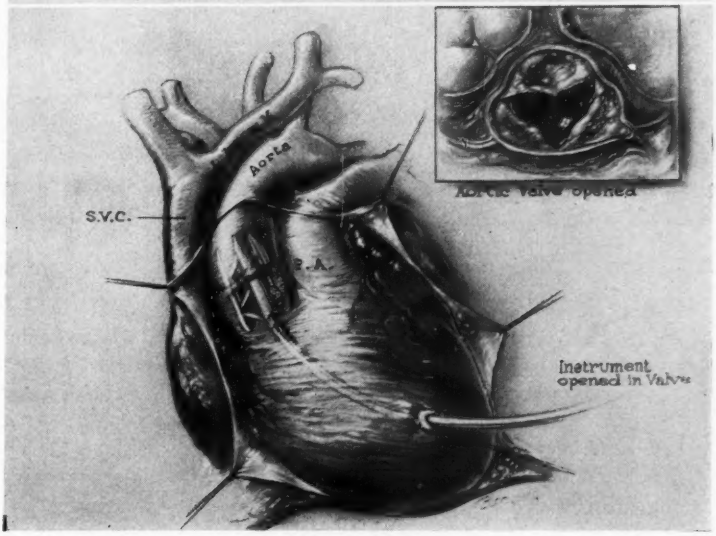


Fig. 4.

Fig. 3.—The valvulotome has been inserted into the stenotic aortic valve through a stab wound in the wall of the left ventricle.

Fig. 4.—Valvulotome in expanded position. The stenotic aortic valve is dilated. Inset, View of aortic valve from above, after dilatation.

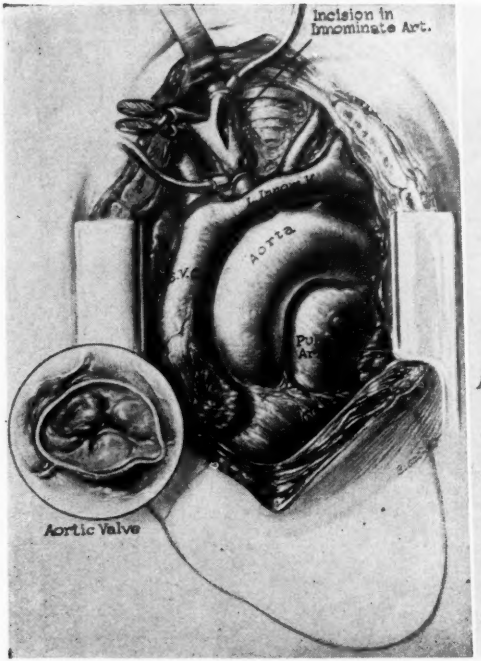


Fig. 5.

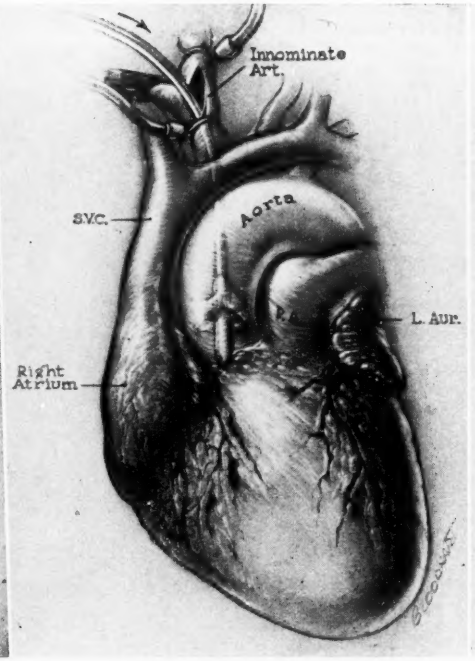


Fig. 6.

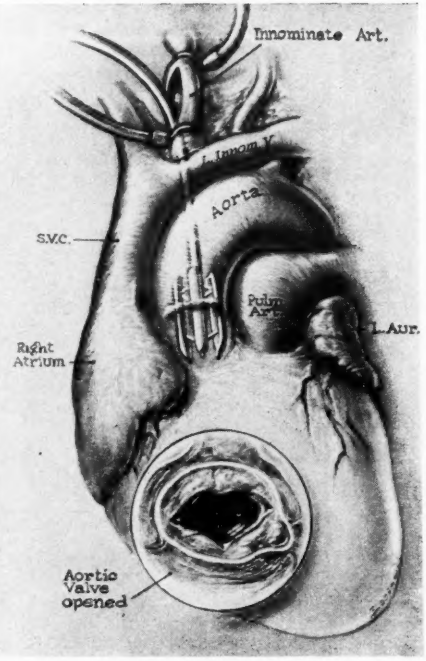


Fig. 7.

Fig. 5.—Exposure of great vessels through median sternotomy incision. The innominate and left carotid and subclavian arteries have been isolated, and an incision is made in the carotid and innominate arteries.

Fig. 6.—The valvulotome has been inserted to the desired level in the aorta through an incision in the innominate artery.

Fig. 7.—The aortic valve is dilated with the valvulotome from the innominate side of the aorta. The inset shows the dilated aortic valve from the innominate side. This procedure did not prove to be feasible in one patient on whom it was attempted.

Bailey[3] and Brock[4] encountered in approaching the valve retrogradely, but thought that exposing the heart through a sternum-splitting incision (Fig. 5), and guiding the end of the dilator by palpating the aortic ring externally would allow the valve orifice to be located and entered without great difficulty (Figs. 6 and 7). Such a procedure was attempted on one patient, but the small aperture of the cone-shaped valve could not be located and the patient expired during the manipulations. Therefore, we returned to the ventricular approach and, since that time, have had no appreciable difficulty using it. It is of great importance to place the mattress suture properly in the ventricular muscle and to emphasize that the assistant should not tighten it excessively, but should partially control bleeding by applying pressure with a small gauze sponge on a hemostat at the point of entry into the ventricle.

When mitral stenosis is present, in addition to aortic stenosis, we open both valves in a single stage procedure. Initially we approached dual lesions through a posterolateral incision in the fifth intercostal space. However, these patients tolerated the supine position much more satisfactorily than the lateral one, and recently we used a slightly longer anterolateral incision in the fourth intercostal space with division of the fourth and fifth costal cartilages adjacent to the sternum. We originally thought that the aortic valve should be opened first in order to allow better coronary artery filling, and also because the concomitant stenotic mitral valve would aid in preventing bleeding from the left ventricle through the ventricular incision by decreasing the amount of blood entering that chamber. However, we now believe that the valve which is most significantly involved should be opened first. For example, if the patient's symptoms are primarily those of angina or syncope, the aortic valve should be opened first, but if they are primarily pulmonary edema, dyspnea, and orthopnea, the initial attack should be on the mitral valve.

RESULTS AND DISCUSSION

A total of 25 patients were operated upon in this series. Of this total, 16 had aortic deformities alone, 8 had mitral valvular deformities in addition to aortic, and 1 had aortic stenosis and a coarctation of the aorta (Table I). Their ages range from 14 to 63 years (Table II).

TABLE I

TYPE OF DEFORMITY	NUMBER
Aortic stenosis	6
Aortic stenosis and insufficiency	10
Total	16
Aortic and mitral stenosis	1
Aortic stenosis; mitral stenosis and insufficiency	3
Aortic stenosis and insufficiency; Mitral stenosis and insufficiency	4
Total	8
Aortic stenosis and coarctation of aorta	1
Total number of patients	25

Of the group with isolated aortic valvular disease, 6 had pure aortic stenosis and 10 had aortic stenosis and insufficiency, as evidenced by a diastolic murmur along the left sternal border. Only 5 of the 16 gave a history of rheumatic fever. Dyspnea and orthopnea were the predominant symptoms in this group, having occurred in 14 of the patients. Angina was present in 11, and syncope occurred in 7. Eight gave a history of having had previous episodes of heart failure (Table III). Cardiac enlargement, to some degree, was present in all these patients, and was primarily due to left ventricular enlargement. Calcification of the aortic valve was found in 13 of the 16 patients at the time of operation, and was determined by contact of the dilating instrument with the calcified valve. Most of these valves could be visualized fluoroscopically prior to operation.

TABLE II.

AGE (YEARS)	AORTIC VALVE DEFORMITY	AORTIC AND MITRAL VALVE DEFORMITY	AORTIC STENOSIS AND COARCTATION OF AORTA
10-20	1	0	0
20-30	1	2	0
30-40	7	2	0
40-50	6	3	1
50-60	0		0
Over 60	1	1	0
Total	16	8	1

In the group of patients with dual deformities, two gave a previous history of rheumatic fever. All histories included dyspnea, and 8 of the 9 noted orthopnea. None experienced syncope, and only 1 had angina (Table III). Seven of the 9, however, had previous episodes of failure, and 4 gave histories of hemoptyses. All of these patients had some degree of cardiac enlargement, and calcified aortic valves were found to be present at the time of operation in 8. Only 1 patient had auricular fibrillation.

TABLE III

	AORTIC VALVE DEFORMITY	AORTIC AND MITRAL VALVE DEFORMITY
Number of cases	16	8
History of rheumatic fever	5	2
Dyspnea	14	8
Orthopnea	14	7
Syncope	7	0
Angina	11	0
Cardiac failure	8	6

There were 4 operative deaths, 2 in the group with aortic disease alone, and 2 in the group with dual deformities (Table IV). The oldest patient in the series, a 63-year-old man with aortic stenosis and insufficiency, died during performance of a retrograde procedure through the innominate artery. The small valve orifice could not be located, and sudden irreversible cardiac arrest occurred shortly after the leading end of the dilator reached the region of the valve. A 31-year-old man with aortic stenosis and insufficiency died of

cardiac failure forty-eight hours postoperatively. Although the valve was widely dilated, the instrument, upon closing, caught a small portion of one of the leaflets between the dilating components and avulsed it, producing severe insufficiency. Immediately following his return to bed, the patient developed severe pulmonary edema, which could not be adequately controlled by massive phlebotomy and supportive medical treatment. The late death in this group occurred in a 49-year-old man, who was judged to have excellent improvement following the operation. However, he received a blow on his head which caused severe cerebral trauma and subsequent death nine months after the operative procedure.

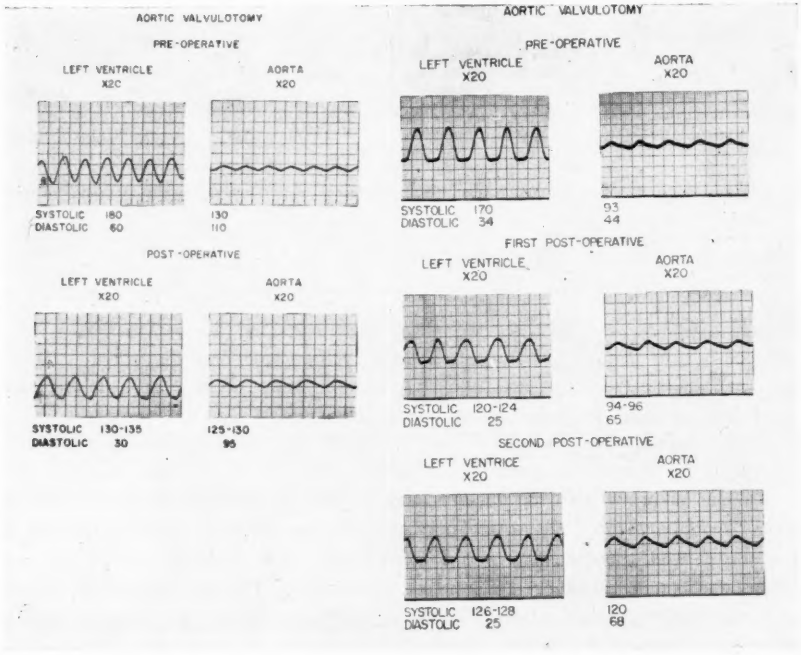
TABLE IV. RESULTS

	SLIGHT IMPROVEMENT	MODERATE IMPROVEMENT	EXCELLENT IMPROVEMENT	OPERATION TOO RECENT	DEATHS	
					OPERATIVE	LATE
Aortic stenosis	1	1	4			
Aortic stenosis and insufficiency		2	3	2	2	1 (Trauma)
Total	1	3	7	2	2	1
Aortic and mitral stenosis			1			
Aortic stenosis, mitral stenosis, and insufficiency			2		1	
Aortic stenosis and insufficiency; mitral stenosis and insufficiency			1	1	1	1
Total	0	0	4	1	2	1
Aortic stenosis and coarctation of aorta			1			
Total results	1	3	12	3	4	2

In the group with dual lesions, one patient, a 42-year-old man, died one week after operation. He apparently sustained a cerebral embolus during the procedure, was disorientated, and exhibited a left hemiparesis of the arm and face when he recovered from the anesthetic. He died suddenly, following a urethral catheterization. Twenty-four hours after operation, a 26-year-old male patient died of a cerebral embolus sustained at the time of operation, without having regained consciousness. Post-mortem examination showed that the valves appeared to have been adequately opened. The late death in this group occurred in a 49-year-old man who died of a pulmonary embolus and severe pulmonary congestion and cardiac failure three months after operation. This patient had extreme cardiac enlargement and a severely calcified aortic valve. The aortic valve had been opened satisfactorily. However, it was necessary to dilate the mitral valve retrogradely by means of the valvulotome through the ventricular incision, because the left auricular appendage was extremely small and almost obliterated by an old organized thrombus. It was thought unwise to consume the necessary time to enter the left auricle through its wall or through a pulmonary vein to perform a digital

mitral commissurotomy. The patient's postoperative course was uneventful and he left the hospital twelve days after operation. He was thought considerably improved, because dyspnea and orthopnea disappeared and his exercise tolerance increased.

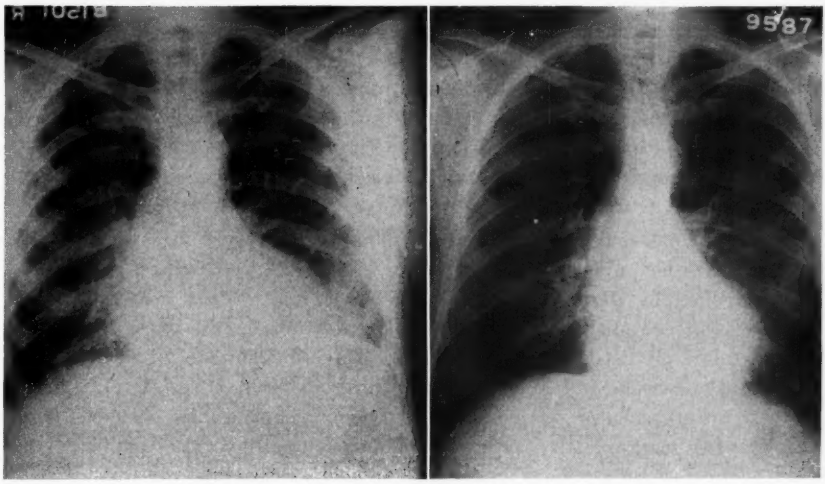
Objective evaluation of these patients in indeed difficult. Criteria include changes in pressures (Fig. 8) measured in some of these patients during operation, changes in heart size (Fig. 9) revealed by roentgenographic examination, and increased exercise tolerance. However, much of the evidence for determining the degree of improvement has been the patient's statement of the disappearance or relief of such symptoms as easy fatigability, palpitation,



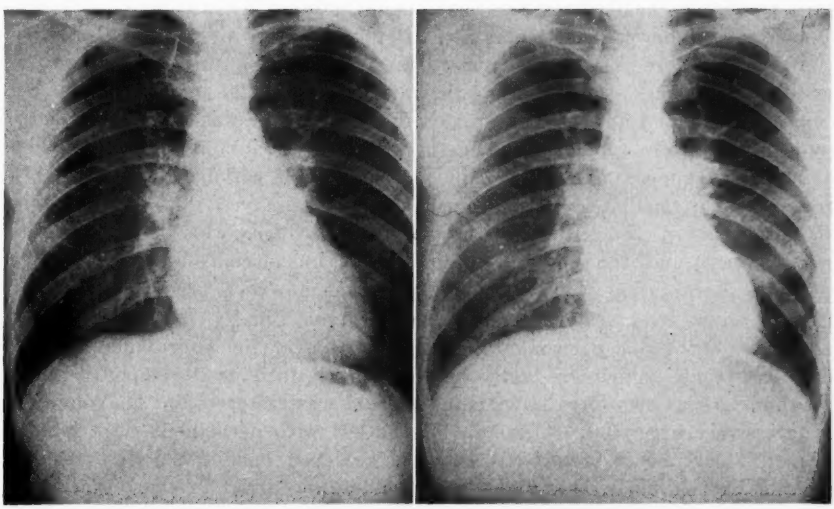
A. *B.*

Fig. 8.—*A*, Pressure tracing from left ventricle and aorta of a 14-year-old boy before and after valvulotomy. The left ventricular pressure is essentially equal to the aortic after the valve is opened. *B*, Pressure tracings from left ventricle and aorta of 44-year-old man before and after valvulotomy. The first postoperative tracing shows valve not adequately opened. The second postoperative tracing shows the systolic pressure in aorta and left ventricle are essentially equal.

exertional dyspnea, and angina, and the patient's ability to resume a gainful occupation. In none of the patients has the murmur disappeared. In some it has become less intense than prior to operation. In several, the diastolic murmur has increased after operation, but in none has it become significant. Heart size has decreased, in some instances, but in others has remained about the same size; in some it has actually become larger.



A. *B.*



C. *D.*

Fig. 9.—*A*, Chest roentgenogram of 47-year-old man with aortic stenosis. *B*, Roentgenogram of same patient three months after aortic valvulotomy. The heart size has become smaller. *C*, Roentgenogram of patient with mitral and aortic stenosis, before operation. *D*, Roentgenogram of patient after operation, demonstrating decrease in heart size.

Of the 19 living patients, the longest follow-up period has been for eighteen months and the shortest for two weeks. Three of these patients, operated on less than two months ago, were not included for evaluation of results. Of those patients with aortic deformities alone, 1 was thought to be slightly improved, 4 showed considerable improvement, and 7 were considered to show excellent improvement. Four of those with mitral and aortic deformities showed excellent improvement. The patient with coarctation of the aorta and aortic stenosis was also considered to show excellent improvement (Table IV).

Severe hemorrhage occurred at the time of operation in three patients. In each case the purse-string suture tore out of the ventricular muscle and was controlled by digital occlusion of the opening while a heavy, mattress suture was placed about it and tied loosely. In another, the purse string tore out, but relatively little bleeding followed. These incidents occurred relatively early in the series, and such difficulty has not been encountered since a mattress suture, placed deep in the ventricular muscle, has been employed with careful manipulation by the assistant controlling it. Cardiac arrest occurred in 5 of the patients, in 4 of whom regular rhythms were restored by means of cardiac massage and administration of drugs. Ventricular fibrillation did not occur in any of the patients. In one patient with aortic stenosis and mitral stenosis and insufficiency, in whom the mitral valve was opened first, a complete heart block took place immediately after the aortic valve was opened. An inherent ventricular rate of approximately 40 began, but cardiac contractions were regular and effective. This rate continued for approximately eight hours, at which time the rate dropped to 20 and the patient became cyanotic and unresponsive. A cardiac pacemaker was employed, whereby electrodes were applied to the chest wall, and contractions were maintained at a rate of approximately 60. The patient became rational and reasonably comfortable immediately. The pacemaker was employed for approximately sixty-four hours. Each time it was discontinued during this period, asystole would occur and the patient would become unconscious after about five seconds. Reinitiation of the stimulating current, however, produced a regular rhythm again with restoration of consciousness. Finally, after about sixty hours, a ventricular pacemaker began to take over for approximately five-minute periods, which gradually became longer until he developed a nodal rhythm. About two days later, sinus rhythm was restored and his condition progressively improved. Although he was left with permanent right bundle branch block, he is considered to have achieved one of the best results. Bundle branch block, one right and one left, occurred in two others, immediately after their aortic valves were opened, but was of no clinical significance. It disappeared in each case after several days. The block in the conduction system in these three patients was probably due either to direct pressure on the atrioventricular node, to the bundle of His itself, or to the production of hemorrhage and edema about these structures. The fact that the changes were reversible seems to confirm this explanation.

In another patient, the flexible shaft which manipulates the dilating components of the instrument broke as the valve was being dilated, but produced no damage to the heart, since it could be easily withdrawn. However, the only dilator available was one which was not so satisfactory as the original one, and the valve was not opened so widely as desired. It is suggested that, because of the complexity of these instruments which makes breakage a possibility, an extra dilator be available during the operation.

Another patient, a 44-year-old man, was found to have a relatively fresh myocardial infarct near the apex of the left ventricle when the pericardium was opened. He had had no symptoms from it, and an electrocardiographic tracing taken immediately after it was discovered failed to demonstrate it. We questioned the advisability of performing the valvulotomy, since the infarct was near the desired point of entry through the left ventricle. However, it was believed that his chances of recovery would be much better, that the likelihood of his developing a ventricular aneurysm would be diminished, and that he would be less apt to have another infarction, because of the increased coronary blood flow if the valve could be opened. Therefore a more medial area on the left ventricle was selected as a point of entry, and the valvulotomy was performed without difficulty. His postoperative course was uneventful and he has shown considerable improvement.

Although the ultimate results in patients operated on by this technique can be determined only after following them for a long period of time, the early results are encouraging.

SUMMARY

During the past few years, experimental and clinical studies have led to the development of a procedure for the treatment of aortic valvular disease. A procedure which consists of introducing a mechanical dilator through a stab wound in the wall of the left ventricle and dilating the aortic valve has been employed in a series of patients. Sixteen patients with aortic deformities, 8 with additional mitral valvular deformities, and 1 with an additional coarctation of the aorta, were operated on in this series. There were 2 operative deaths and 1 late death in the group with isolated aortic valvular disease, and 2 operative and 1 late death in the group with dual deformities. Technical considerations and problems encountered in treating this series of patients are discussed.

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DISCUSSION

DR. CHARLES P. BAILEY, Philadelphia, Pa.—We have performed a total of 205 operations for aortic stenosis, 11 by a now obsolete technique, 173 by the ventricular route with the standard dilator, and the remaining ones by the transaortic route. You will note the greater mortality experienced when these operations were done by the ventricular route than when operated on by the supravalvular approach route. In this group of cases you will note that the condition is essentially isolated aortic disease with predominant stenosis and perhaps some insignificant insufficiency. You will note that the operative mortality is about 25 per cent, even if we exclude those with dynamic aortic insufficiency. In the group in which there was associated mitral disease and in which both valves were operated on simultaneously, the operative mortality was significantly lower (except in true multivalvular conditions), particularly in those with only a combination of aortic stenosis and mitral stenosis. Even though this series included all types of cases in various stages of advancement, the high mortality led us to feel that surgery by the transventricular route inherently carried a high risk. Approximately 40 per cent of our mortality was due to ventricular fibrillation during surgery.

We have operated on 21 cases, and the mortality has been 14 per cent. I think that improvement in statistics was due not only to the avoidance of ventricular fibrillation but also to the avoidance of production of aortic regurgitation. This latter complication can inadvertently be accomplished with the dilating instrument. The aorta may be entered by pinching off the aortic wall with the help of a curved Potts toothed clamp or a Satinsky clamp of a special type, and sewing a pouch of pericardium to an opening made in the excluded portion of the aortic wall. A purse-string suture is placed around the periphery of the patch to maintain hemostasis. Then a finger is inserted through the pouch, into the aorta, and into the valve. In more than one-half of these cases, almost exactly the same percentage as in mitral stenosis, an adequate commissural separation can be obtained by digital pressure, perhaps reinforced by counterpressure with the other hand from the outside of the aorta. If necessary, one can slide a mitral guillotine knife along the finger to cut a resistant commissure. However, we have devised this instrument which has only two expanding blades, which is worn on the finger for forceful splitting of the commissures.

One can follow the expansion of the blades by the finger during the time the valve is being split. Then if the third commissure is to be separated, the instrument is turned 120 degrees and is expanded with digital guidance. There is one other advantage in operating on these patients from above; that is, if there is coexisting aortic insufficiency or if aortic insufficiency is created inadvertently, it may be treated immediately. A threaded probe is passed along the intra-aortic finger, and punctures the aortic wall just to the right of the anterior commissure. A ball of nylon with a tail is attached to the portion of the suture that comes out of the pouch. The ball is drawn into the aortic lumen until the finger tip recognizes its function. By this method one can usually cut off all the regurgitation.

After completion of the aortic commissurotomy in one case the pressure was 138/68. However, there was a definite diastolic thrill palpable over the outflow tract of the left ventricle and the finger could recognize a central gap in the closure of the valve. When we put in a ball, the diastolic pressure rose to 90 and the diastolic thrill disappeared.

DR. ROBERT P. GLOVER, Philadelphia, Pa.—Of the four cardiac valves with which the cardiovascular surgeon must deal, the aortic valve is and will continue to be, in my opinion, the most difficult to treat surgically because of its position in the vascular stream.

It is the last valve traversed by the blood stream as it courses through the heart. Thus all cardiac chambers lie proximal to it and share in the strain and stress occasioned by the dysfunction of this valve. When evaluating a patient with aortic stenosis for surgery it it paramount to recognize this anatomic location, for it means that the *left ventricle* has absorbed and sustained the greatest myocardial damage. In this respect it differs from the evaluation of a patient with mitral stenosis, for here the myocardial damage has, for the most part, been proximal to the valve in the left atrium and right heart, with a reasonably normal left ventricle in front to take over effective cardiac output once the mitral valve has been opened. All cardiac surgical procedures are doomed to failure if the *keystone of the heart,* the left ventricle, is ineffective. A high percentage of mitral stenosis cases that have been in congestive failure (pulmonary and right heart) can be salvaged, but clinical failure of the same degree secondary to aortic stenosis is fraught with a high mortality and a poor clinical response.

We have divided the clinical course of the patient with aortic stenosis into five stages: Stage I, the murmur without symptoms; Stage II, the onset of vague precordial distress, and palpitations—general heart consciousness; Stage III, the development of syncopal attacks and/or angina. To this point the left ventricular myocardium has not failed and surgery performed (preferably during stage II) during this time carries a reasonable operative mortality (8 per cent) and a favorable result in almost 75 per cent of cases. On the other hand, once the integrity of the left ventricular myocardium has disintegrated as in Stage IV—episodes of pulmonary congestion, or Stage V—frank congestive failure, the mortality rises to 44 per cent and 85 per cent, respectively, and the result obtained in the survivors is gradually reduced to a negligible one. These factors are true regardless of the technique of aortic commissurotomy, whether performed through the left ventricle or the aorta.

DR. DWIGHT E. HARKEN, Boston, Mass.—I am anxious to say something about the objective studies and measurements relevant to the surgical correction of aortic stenosis. Dr. Muller is to be congratulated on his work.

First, pure aortic stenosis producing syncope, angina, and/or left ventricular failure is a malignant phase in the life cycle of this disease. Of the 26 patients in our series that did not accept surgery, or who procrastinated for one reason or another, 23 were dead within four months. This is very different from *combined* aortic and mitral stenosis. Combined involvement represents a wider clinical spectrum, the clinical evaluations of the results of surgery are quite different, and technically the surgery is much easier. When we talk about aortic stenosis we should leave combined aortic and mitral stenotic lesions out of the series.

Second, I should like to simplify this problem further by dividing aortic stenosis into three syndromes that we recognize. All three have the murmur of aortic stenosis in the aortic area transmitted to the neck, calcification in the region of the aortic valve, left ventricular hypertrophy, and more or less flattening of the brachial tracing. Even with these features indicating so-called pure aortic stenosis we can make out different patterns. The first syndrome is recognized at fluoroscopy, for the very large left ventricle and the aorta move very little. In all probability this very large left ventricle is associated with a small, hypertensive chamber and a small stroke volume that accounts for the lack of motion. This is the true aortic stenosis that we believe has the highest gradient across the valve and most urgently needs direct surgical therapy. Certainly it is urgently indicated if the patient has syncope, angina, and/or left ventricular failure. The second syndrome looks exactly the same on the roentgenograms, has all the above qualifying features of so-called pure aortic stenosis in auscultation, calcification, left ventricular hypertrophy, and symptomatology. There may even be flattening in the brachial tracing if there is any degree of left ventricular failure. However, fluoroscopy reveals a very active left ventricle and aorta. This is aortic stenosis and insufficiency with slight ventricular failure. The pulse pressure may not be widened. This lesion

does not respond as well to the surgical correction of stenosis. Insufficiency cannot be quantitatively assessed by the diastolic murmur. The third syndrome again presents the same basic qualifying features with the murmur, calcification, left ventricular hypertrophy, and flattening of the brachial tracing. Here the enlargement is not so marked as that of the first two syndromes, and the presenting symptom is generally that of angina. In this situation something has caused the development of angina before the usual marked left ventricular hypertrophy has taken place. Here coronary insufficiency due to atherosclerosis is associated with incidental and perhaps insignificant aortic stenosis. Two of the 9 deaths in 18 operations for aortic stenosis have been due to coronary occlusion in this group. More objective studies at the operating table might have directed our attention to operations that relieve angina without correcting the aortic stenosis, namely, phenol de-epicardialization, poudrage, and pneumonopexy.

A few words about the objective studies in combined and pure stenosis may be in order. When a patient with aortic and mitral stenosis is considered for surgery, a brachial tracing is taken. If there is no prolongation of the upstroke or total ejection time, we assume that no hemodynamically significant aortic stenosis exists. If the ejection and upstroke time is prolonged, we have to decide whether it is due to block in the aortic valve, alteration in peripheral capillary resistance, left ventricular failure, or a low cardiac output. Very often this proves that the patient does not need combined aortic and mitral surgery. If such a decision cannot be made from clinical evaluation and brachial tracings, we measure the pressure gradient across the aortic valve at the operating table before the mitral stenosis is corrected. The gradient is determined by placing needles in the base of the aorta and left ventricle. The Sanborn manometer measures pressures, and these are recorded by the Polyviso writer. If this shows that the gradient is less than 50 mm. Hg when there is presumably a good cardiac output, we do not operate on the aortic stenosis. Our feeling is that at the present stage in the technique of correcting aortic stenosis, the risk of aortic valvulotomy is greater under these conditions than the potential benefits. More than 30 patients have been eliminated from surgical intervention by this technique. These patients have been followed for more than four years, and we have no reason yet to regret this decision. In other words, the aortic stenosis has not become clinically significant. These patients have been spared an extra risk that would presumably not have benefited them.

A similar type of measurement is also carried out routinely in our operations which is now defined as pure aortic stenosis (the first group of the three syndromes mentioned previously). Such gradients may assess the effect of our correction of stenosis. Our valvulotome, producing fracture and dilatation, has completely eliminated the gradient in the most favorable cases. Ordinarily the gradient is lowered from 80 or 100 mm. Hg to 30 or 50 mm. Hg.

It is too bad that we have not yet developed really satisfactory techniques for the preoperative evaluation of the degree of aortic stenosis. It is also unfortunate that moment-to-moment determinations of cardiac output are not available. Output determinations would give a third dimension that is so important to our present pressure gradients at the operating table. I hope that we will soon have these available, and then a great step forward in the objective evaluation will be realized.

As to the possibility of preoperative evaluation of the pressure gradient across the aortic valve, I can only say that retrograde catheterization down the aorta has not been satisfactory. We have, however, been working with transbronchoscopic left heart catheterization and have carried it out twelve times without complication. As yet we have not been able to control our catheter satisfactorily, and have had difficulty in getting it to pass through the needle carrier (which goes from the bronchus into the auricle) into the auricle, then to the ventricle, and out through the aorta. When we are able to control this catheter in this course, we may be able to have a very simple diagnostic test that may spare a large number of patients some very major surgery.

These attempts at objective study of course sort out the very worst patients for surgical intervention. This unquestionably accounts for our combined early and late mortality rate of 50 per cent. On the other hand, it has properly spared a substantial number of patients a major operation.

I hope others of this group share my interest in the objective evaluation of techniques and diagnoses. Such objectivity will improve the surgery for aortic stenosis.

DR. EGBERT H. FELL, Chicago, Ill.—It is evident that there is no ideal treatment for aortic stenosis. The procedure to be presented is not ideal either, but has some virtues, I believe, that the transventricular approach does not have. Our experience is not great; it consists of dilating the aortic valves of eight patients varying in age from 3 months to 50 years.

Our approach to the aortic valve is through the sternal splitting incision exposing the ascending aorta and heart anteriorly. The aorta is freed from the mediastinal structures so that the aortic valve may be palpated carefully. The operator, standing on the left side of the patient, grasps the aorta gently, at the level of the aortic valve, between the index finger and the thumb of the left hand. The right hand guides without force the closed dilator through the stenosed valve opening into the left ventricular chamber. To date no difficulty has been experienced in passing this strong, small instrument. No force should be used and at systole when the valve is at its greatest dimension the dilator has literally dropped into the ventricle. The dilator is then opened and brought back, gradually enlarging the stenotic opening. The left index finger and thumb maintain their position at the level of the aortic valve, and the operator can easily feel dilation as it takes place if the instrument is opened sufficiently for adequate dilation.

We have not experienced, and I believe these are some of the virtues of this procedure, cardiac irregularity, cardiac arrest, ventricular fibrillation, or uncontrolled hemorrhage.

We use a strong, small instrument calibrated to 24 mm. It can be opened in varied positions in the aortic valve opening. We usually slide it very gently into the orifice and down into the ventricle. It is then brought back and opened up completely to 24 mm. Dilation is repeated in the same or varied positions until the operator believes adequate dilation has resulted. The systolic blood pressure improves and a diffuse thrill is felt in the aorta instead of the localized thrill caused by the fine jet of blood striking the aortic wall, which preceded dilation.

One patient died a week after surgery of multiple pulmonary emboli. We have a photograph showing the well-healed incision which was made in the aorta for insertion of the dilator. The opening of the stenosed valve of this patient as seen from the aortic side during diastole is very small.

A photograph of the same valve propped open as it would be in systole shows that dilation was well accomplished along the commissures. This patient had marked improvement of blood pressure immediately following dilation.

DR. H. BRODIE STEPHENS, San Francisco, Calif.—I have not had the opportunity to apply the method I am about to describe, but it appears to me that this maneuver may have practical application.

If one is faced with uncontrollable bleeding from one of the chambers of the heart, the heart can be delivered out of the pericardial sac and the two cavae temporarily occluded. This sudden inflow occlusion will immediately lower the pressure within the heart and render it possible to insert and tie sutures or apply clamps, depending upon the source of bleeding.

At the present time I believe this is the procedure of choice for dilation of a stenotic aortic valve.

DR. EDGAR W. DAVIS, Washington, D. C.—I was very much in sympathy yesterday with Dr. Ralph Adams, who brought up the problem of what to do for his patient who fibrillated for one hour and fifty minutes. I wish now to pose a question about a problem which unfortunately does not allow that amount of time. I believe we all realize that the closure of a wound in the left ventricle, made for aortic stenosis, is much more difficult than the closure of any other cardiac incision. The muscles are very spongy and friable and sometimes the sutures, when tied just barely tight enough to arrest the spurting of blood, begin to cut through the muscle. It is similar to trying to sew together two pieces of cheese. Recently we encountered a case like this in which we successfully opened the aortic valve but were unable to close the wound when the instrument was withdrawn. Various sizes of suture material were used, with very deep bites taken into the ventricular wall, but the blood continued to flow and the patient died on the table. Needless to say, the usual precautions were taken to prevent this bleeding. I am wondering if some of the experts can tell me how closure can always be effected.

DR. CHARLES P. BAILEY, Philadelphia, Pa.—There are only two answers to the problem of a friable left ventricular wall if you go through the ventricle. That is one of the inherent problems in aortic stenosis surgery. The ventricle becomes thick and soft and may not hold sutures.

One method of suturing has been devised by Dr. Ray Rumel, of Salt Lake City, Utah. He has developed a clamp which has several prongs on each jaw. This is placed in the myocardium on each side of the wound and holds the wound together, giving all the time necessary to place hemostatic mattress sutures. However, even that method of suturing fails if the texture of the heart is comparable to a cheese, and a pulsating cheese at that. In these latter cases the only way we have been able to stop bleeding is by the use of a suggestion made by Dr. Jerome Head, who saw me lose one such patient. If two segments of a removed rib are drilled at either extremity one can pass a large threaded curved needle through a hole in one and under the ventricular incision. The emerging needle is passed through a hole in the other bone fragment, is returned through the other opening in this fragment, passed through the ventricle under the tamponading finger and finally penetrates the original bone fragment at the opposite end. When tied, this amounts to a huge approximating mattress suture, using the bone fragments to convert it into a retention suture. In three cases where this technique was applied hemostasis was achieved in each.

DR. FELIX HUGHES, Memphis, Tenn.—Dr. John Kehne has been doing some work in the Research Laboratory at Kennedy Hospital that might apply in this instance. He has taken portions of fascia, excised portions of the aorta of a dog, and has sewn this fascia in as a patch. The patch becomes endothelized and smooth, and I am sure it could be used in the aorta. In the heart, with the coronary in between, it might not work, but it certainly works in the aorta of dogs. Some of these dogs have been followed for six and eight months.

DR. JULIAN JOHNSON, Philadelphia, Pa.—It may be that the ventricular wall bleeding which has scared me to death has not been quite as bad as the cases Dr. Bailey has had. I have not used any ribs, but in several instances in which bleeding was severe we have been able to stop it by putting Oxycel gauze under the sutures and tying over it. We have had no cause to regret this. I have never liked to put foreign material in any wound but we have had at least two, perhaps three, cases in which I thought we could not stop the bleeding from the ventricular wound, but were able to tie the sutures over the Oxycel gauze.

DR. WILLIAM H. MULLER, Jr. (Closing).—I wish to thank all the discussants very much. I am not going to try to compare the ventricular approach with the retrograde approach which Dr. Bailey has used, having had no experience with the latter. There are virtues for both, but in our own hands, we prefer the ventricular approach at

present. As for the nylon ball which he demonstrated, something of that sort is needed to prevent regurgitation at the time of the operative procedure in patients who have significant aortic insufficiency before operation. However, I wonder if such a prosthesis might break off after a long period and lodge as a saddle embolus later, since it is attached by only one nylon suture.

As for the control of bleeding from the ventricular wound, we originally had considerable difficulty with it, so much so that we tried a retrograde approach through the innominate artery. In order to prevent bleeding we think one should place the pulse-string suture as deeply as possible and leave as much of the suture exposed between bites as is buried. Also, the assistant should not tighten the purse string excessively but should aid in controlling bleeding from the wound with a small gauze sponge on a long hemostat. By following these principles we have had no difficulty with bleeding from the ventricular wound recently.

LOBAR AGENESIS OF THE LUNG

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INTEREST in congenital anomalies of the pulmonary system has been heightened in recent years by the striking advances which have been made in thoracic surgical and diagnostic techniques. As a result, lesions of this type are being described with increasing frequency. Lobar agenesis of one lung associated with a congenital abnormality of the remaining bronchial tree on that side is an exceptionally rare developmental defect. This report concerns such a case in which the correct diagnosis was made preoperatively on the basis of the physical findings, chest roentgenograms, bronchoscopy, bronchography, and pulmonary function studies. The diagnosis was confirmed at operation and further varified by histologic study of the resected specimen.

The literature contains reports of only four cases which could be representative of lobar agenesis of the lung. Morton, Klassen, and Baxter[1] reported two cases; one was proved at operation, and the other at necropsy. In each patient there was agenesis of the right upper and middle lobes. Field[2] recorded a case in which the lingula was absent, the left lung small, and the main-stem bronchus long and directed posteriorly. Ferguson and Neuhauser[3] described a patient who was found on bronchoscopic examination to have one communicating bronchus to the right upper lobe with an absence of the right middle and lower lobes. The latter two patients were not operated upon, and neither came to autopsy. These cases, therefore, lack definite anatomic proof of lobar agenesis.

CASE REPORT

R. E. H., Reg. No. 1108288, a 21-year-old white man, was admitted to the United States Naval Hospital, St. Albans, New York, on March 1, 1953, complaining of a mild cough, moderate shortness of breath, and a pronounced wheeze over the left chest. Shortness of breath had been present for several years, but it had steadily increased in severity during the previous two years. The wheeze was first noted about two years previously, and had become progressively worse. The patient had had a chronic cough for approximately eighteen months and raised small quantities of fairly thick yellowish sputum. There were no other complaints. The *past history* revealed nothing of significance. He had not been hospitalized previously and there had been no serious illnesses, injuries, or operations. His *family history* was not remarkable. General *physical examination* showed normal findings except for the chest. The left hemithorax was definitely diminished in size in comparison with the right. Expansion on the left was markedly decreased. Percussion revealed hyperresonance throughout the entire left chest. The area of cardiac dullness was shifted to the left. On auscultation, it was found that the breath sounds were distant and diminished on the left, and normal on the right.

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It was clear that the patient was not moving air well on the left. No râles were present but a loud wheeze could be heard on the left. It was most pronounced in the hilar region, present on both inspiration and expiration, but accentuated on expiration. Chest *fluoroscopy* served to confirm the physical findings. There was increased radiolucency of the left lung field, suggesting emphysema. The right lung appeared normal. The heart was displaced slightly to the left of the usual position. The mediastinum shifted rather sharply to the right on expiration and returned to the left on inspiration, demonstrating trapping of air in the left lung. This lung remained abnormally radiolucent in all phases of respiration. The bronchovascular markings were much diminished on this side. *Chest roentgenograms* (Fig. 1) verified the fluoroscopic findings, but added no additional worth-while information. *Bronchoscopy* showed a normal larynx and trachea. The main carina was sharp, vertical, mobile, and in the midline. There were no abnormalities in the right bronchial tree. The left main-stem bronchus was somewhat longer than usual. There was a fusiform narrowing on this bronchus, its caliber diminishing distally, so that its shape was that of a slender, elongated cone or funnel. The tip of the 8-mm. bronchoscope would barely enter the left stem bronchus, and

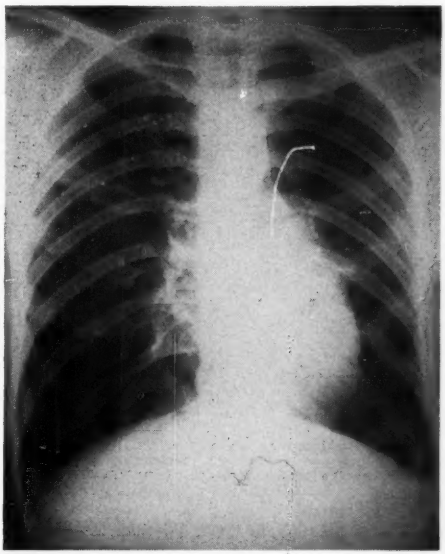

Fig. 1.—Posteroanterior chest roentgenogram showing increased radiolucency of the left lung, with decreased bronchovascular marking and shift of the cardiac shadow to the left.

the stenosis was so marked that a 3-mm. suction tip would not pass a point just proximal to its first bifurcation, 6 cm. from the main carina. The mucosa appeared to be healthy. No mucosal furrows or folds were present and there was no evidence of active or healed endobronchial disease. It was therefore concluded that the bronchial abnormality was the result of a developmental defect and that the bronchus was not constricted because of extrinsic pressure, or stenosed as the result of intrinsic bronchial pathology. Although the bronchoscope would scarcely enter the left stem bronchus, two carinae and three small bronchial orifices could be distinguished in the distance. These three orifices were arranged in a row from left to right, the most medial arising at a slightly higher level than the remaining two. Except for the pronounced narrowing of all visible bronchi, the bronchoscopic appearance was that which might be expected if the lower lobe were absent and the upper lobe rotated downward. Nothing suggestive of a pre-existing lower lobe bronchus was visualized, despite

careful observation. *Bronchograms* graphically demonstrated the findings observed at bronchoscopy (Figs. 2 and 3). The trachea and right bronchial tree were normal. There was marked stenosis of the left stem bronchus with progressively increasing diminution in caliber in a distal direction, so that at its bifurcation, the bronchus was no more than 2 or 3 mm. in diameter. This bronchus ended in a trifurcation, the most medial branch originating slightly higher than the middle and lateral bronchi, which began at a common level. Stereoscopic frontal (Fig. 2), oblique projections (Fig. 3), and a true lateral film showed that the most lateral bronchus coursed upward toward the apex and represented the apical-posterior segment

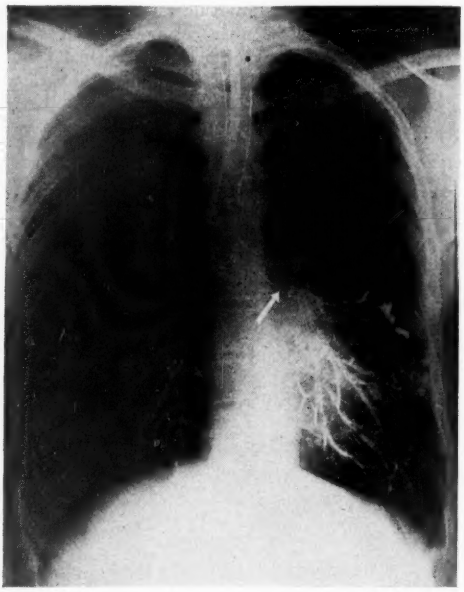

Fig. 2.—Posteroanterior bronchogram showing stenosis of the left main-stem bronchus, absence of a lower lobe bronchus, and excellent visualization of the three segmental bronchi of the left upper lobe. There is marked cylindrical bronchiectasis of the anterior segment of the left upper lobe.

of the left upper lobe. The middle of the three bronchi passed anteriorly and represented the anterior segmental bronchus. There was cylindrical bronchiectasis of this segment. The remaining bronchus, the most medial and the highest in origin, was the lingula, and this bronchopulmonary segment filled the lower portion of the left chest, the inferior lingular segment lying posteriorly, and the superior segment anteriorly. Despite excellent filling with contrast medium, no trace of a lower lobe bronchus could be detected.

Cardiopulmonary function studies revealed the following pertinent findings:

Maximum breathing capacity: 108 L. per minute (74 per cent of predicted)

Timed vital capacity:

Total vital capacity—5,200 ml., first second—48 per cent, second second—63 per cent, third second—78 per cent (expected normal percentages: 75, 90, and 97 per cent for the first, second, and third seconds, respectively).

Bronchospirometric data (normal ranges given in parenthesis):

O_2 uptake—right 86 per cent (52 to 58 per cent), left 14 per cent (42 to 48 per cent); minute ventilation—right 88 per cent (52 to 58 per cent), left 12 per cent (42 to 48 per cent); vital capacity—right 82 per cent (52 to 58 per cent),

left 18 per cent (42 to 48 per cent); residual volume 2,150 ml. (181 per cent of predicted). These findings were interpreted as being indicative of obstructive breathing.

Cardiac catheterization showed the following pulmonary artery pressures: 21/15 and 26/13 on two examinations at rest, the mean pressures being 19 and 18 mm. Hg, respectively. On exercise while breathing room air, the pressures were 25/15 (mean 20) and on exercise breathing a low oxygen mixture, they were 32/18 (mean 29). The brachial artery pressures were 106/71 (mean 96) at rest, and 131/80 (mean 101) on exercise. The arterial oxygen saturation was 95 per cent at rest, 96.9 per cent on exercise breathing ambient air, and 81.2 per cent with exercise plus hypoxia.

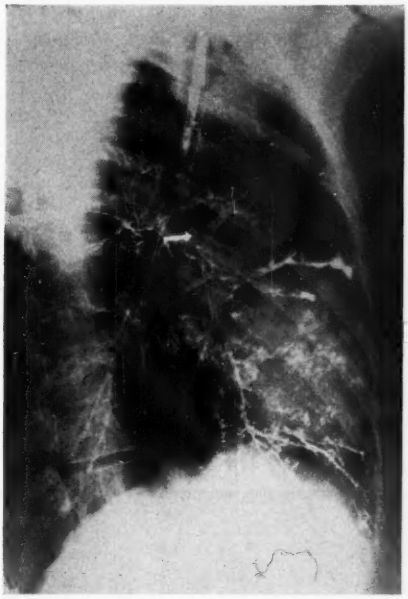

Fig. 3.—Right anterior oblique bronchogram, which shows the stenotic left main-stem bronchus to better advantage. The three segmental bronchi are well visualized, and the bronchiectasis of the anterior segment is again delineated. Note complete absence of lower lobe bronchi.

Left thoracotomy was performed on March 19, 1953. The preoperative diagnosis was lobar agenesis, left lower lobe, and congenital stenosis, left main-stem bronchus. When the chest was explored, a completely free pleural space was found. An emphysematous left upper lobe, showing little anthracotic pigmentation, completely filled the left hemithorax. There was no fissure. A small meaty structure, about the size and shape of a man's thumb, was attached to the diaphragmatic surface of the left upper lobe. This structure was not air containing. It was considered to be a vestigial remnant of the left lower lobe. The operative plan was to resect the left main-stem bronchus and reanastamose that structure, at its bifurcation into the upper lobe branches, to the trachea at the main carina. Accordingly, the anterior pulmonary vein, the pulmonary artery, and the left stem bronchus were dissected out, the vascular structures retracted, and the bronchus was divided near the main carina proximally and again just proximal to its bifurcation distally. The bronchial lumen, which measured 2 to 3 mm. in diameter at its bifurcation, was considered inadequate to permit a

Fig. 4.—Surgical specimen inflated with room air, and with the main-stem bronchus occluded with a hemostat. The left upper lobe has assumed the configuration of the left lung. The oblong dark area just below and to the left of the center of the specimen is the vestigial remnant of the undeveloped lower lobe. It had no bronchial communication.

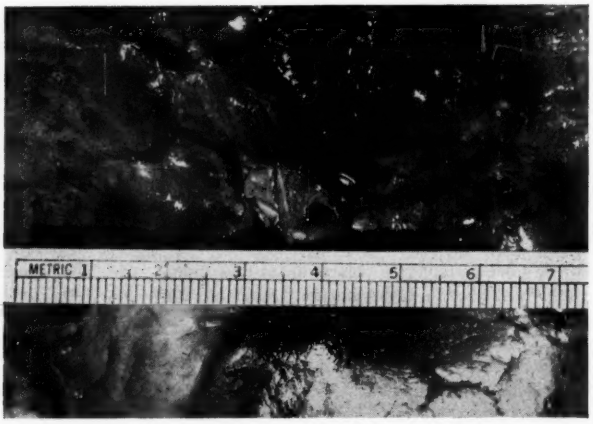

Fig. 5.—End-on view of the left main-stem bronchus where it was severed near the main carina. This bronchus measured 4.5 mm. in diameter at its widest point.

satisfactory anastamosis. Left pneumonectomy was therefore performed. The inferior pulmonary vein was represented by an atretic, fibrous, cordlike structure. In view of the proved bronchiectasis of the anterior segment, the marked emphysema of the left upper lobe, and the very poor pulmonary function on the left, as shown by bronchospirometry, the decision to perform a total resection on the left was probably a wise one, even if resection of the stenotic left stem bronchus with reanastamosis had proved to be technically feasible.

Examination of the gross specimen, inflated with room air, showed a left upper lobe the size and shape of a lung (Fig. 4). Attached to the base of this lobe was a small sliver of nonair-containing tissue resembling liver substance. The left main-stem bronchus measured 4.5 mm. in its greatest diameter (Fig. 5). Sections of this bronchus showed completely normal histology. The upper lobe bronchus gave rise to the usual three segmental bronchi. There was no lower lobe bronchus. Histologic examination showed bronchiectasis of the anterior segment of the left upper lobe and generalized emphysema.

The patient's postoperative course was uneventful. Postoperative pulmonary function studies carried out two weeks after pneumonectomy revealed no significant findings except a reduction in the residual volume to 1,565 ml. (132 per cent of predicted). A seven-rib tailoring thoracoplasty was performed on April 9, 1953.

Pulmonary function studies on May 9, 1953, showed the residual volume to be reduced to 985 ml. (83 per cent of predicted).

Cardiac catheterization at this time showed normal pulmonary artery pressures: 18/6 (mean 13) and 18/11 (mean 16) on two examinations at rest, 23/10 (mean 19) on exercise breathing room air, and 26/14 (mean 23) on exercise with hypoxia.

Oxygen saturation was 97.6 at rest, 96.4 on exercise breathing ambient air, and 80.3 on exercise breathing a low-oxygen mixture.

Postoperatively, the patient's clinical condition was good. The wheeze had disappeared. He had no cough or expectoration. His shortness of breath was unchanged.

DISCUSSION

The etiology of lobar agenesis appears to be a developmental error of endogenous origin which is closely related to that of agenesis of a lung but has its origin in a correspondingly later period. Arey[4] has described the embryonic development of the respiratory system as beginning with a groovelike evagination, arising on the ventral side of the esophagus in the 3 mm. stage, known as the laryngeotracheal groove. At the 4 mm. stage the two lung buds grow out from the posterior end of this groove. Later in the development, the anlage of the trachea and esophagus become separated by a constriction, interrupted at the cephalic end by the larynx, which can be seen in the 5 mm. embryo. At the 7 mm. stage, the lung buds give rise to two lateral bronchi on the right, and one lateral bronchus on the left. The upper end of the right bronchus is destined to be the apical bronchus, the lateral bud to be the axis of the middle lobe, and the termination of the main-stem bronchus to be the lower lobe. On the left side, the lateral bud forms the upper lobe bronchus and the main-stem bronchus, the lower lobe. In later development, these hollow evaginations grow out into an envelope of connective tissue, enlarge rapidly, and branch into a true treelike tubular system. On the fine terminal tubules arise small outgrowths which constitute the alveoli. This description of development suggests that lobar agenesis occurs between the 5 mm. and 7 mm. stage of embryonic development in which there is either complete absence of the lateral buds or of development of the main-stem bronchial buds.

Most authors have accepted Schneider's classification[5] of agenesis of lung which was established in 1912 as:

1. True aplasia of the lung and bronchus in which there is no trace of a bronchus.
2. Aplasia of the lung in which the bronchus is represented by a blind pouch or a nodule of cartilage and fibrous tissue.
3. Extreme hypoplasia of the lung in which the main bronchus is normal in size and shape and ends in a fleshy structure.

The great majority of cases of agenesis of the lung represent post-mortem findings, and the correct diagnosis of this disorder during life is exceptional. Garber[6] has suggested that the total of proved cases does not exceed 100. Wexels[7] has recorded eighty-seven cases, of which twenty-eight were diagnosed during life; thirteen of the latter were verified by autopsy, and fifteen of the patients were still alive at the time of his report.

The collected literature[8-15] indicates that agenesis of the lung is three times more frequent on the left than on the right. It is more common in males than in females in the ratio of 3:2. Most patients with this disease die early in infancy or childhood from either respiratory infection or as a consequence of other congenital anomalies. Maier and Gould[16] have suggested vascular compression of the airway by an anomalous course of the pulmonary vessels as the cause of death in some of these cases. This group of anomalies does not invariably result in early death. Valle and Graham[17] reported a case of agenesis in a 41-year-old woman and have recorded other patients 58, 65, and 72 years of age, respectively, with this developmental defect. The large majority of cases reviewed showed various degrees of hypoplasia with little or no functional lung tissue on the affected side.

A variety of congenital anomalies have accompanied agenesis of the lung. Lucas, Dotter and Steinberg[18] reported a patient who had a patent ductus arteriosus and agenesis of the lung with reversal of the blood flow. Other associated defects have been: absence of ipsilateral pulmonary vessels, absence of the diaphragm, anal stricture, tracheoesophageal fistula, accessory thymus, hypertrophic thymus, diaphragmatic hernia, absence of a vagus nerve, cleft palate, agenesis of the spleen, kidney, and ureter, esophageal atresia, other cardiovascular anomalies, hypoplasia of the face, and incomplete lobulation of the remaining lung. In some instances, three bronchi were supplying a unilobar lung.

SUMMARY

Although developmental anomalies of the respiratory apparatus are not exceedingly uncommon, lobar agenesis of the lung is a rare lesion. A case of agenesis of the left lower lobe and congenital stenosis of the left main-stem bronchus is reported here. This appears to be the third such case to be anatomically proved, the second to be confirmed at operation, and the first in which the correct diagnosis was established preoperatively. These interesting anomalies are clinically unimportant unless they give rise to symptoms or cause

functional deficits. When either of these situations prevails, surgical treatment is usually indicated. While it was not feasible in the case reported herein, reconstructive surgery may be possible in some patients with this or a similar type of defect.

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A SIMPLE METHOD FOR DOING A THORACENTESIS USING A PLASMA-COLLECTING VACUUM BOTTLE

HORACE A. ALBERTSON, M.D., DANIEL LEAVITT, M.D., AND JOHN R. GAMBLE, M.D.
ROANOKE, VA.

IN 1951 Beech wrote an article advocating the use of the blood donor set as a practical system for thoracentesis. We have found his method a highly effective one for removal of pleural effusions. We have only modified the method slightly. Because of its extreme simplicity, we believe that it should once again be brought to the attention of those who may have missed Beech's original article.

Beech used the 600 c.c. vacuum flask, tubing, and needles that are used for obtaining donor blood. Because of its inexpensiveness and size we have utilized the 1,000 c.c. vacuum bottle ordinarily used in the collection of plasma containing no citrate or anticoagulant,* plus a plastic disposable blood donor set. This donor set has a needle 1½ inches long to be inserted into the pleural cavity. (Fig. 1.) In the great majority of patients a needle of this length is quite sufficient. If, due to thickness of the chest wall, a needle of greater length is needed, then a very simple arrangement using two glass needle adapters attached to a section of fairly stiff rubber tubing can be sterilized and needles for any desired length used. In the rare case in which this is necessary, one must use considerable caution in inserting the needle attached to the glass needle adapter into the vacuum bottle. This is best done by holding the needle near its hub rather than holding the glass needle adapter. It is rare, though, that the thickness of the chest wall exceeds 1½ inches, and it has been our experience that this is a good safeguard and will prevent one from probing too deep into the chest and damaging the underlying lung tissue.

Thoracentesis, as we perform it, consists of preparing the chest with an antiseptic and inserting a small amount of Novocain under the skin. Next, the tubing is clamped with a hemostat and one of the needles is inserted through the hard rubber cap of the flask. The other needle is inserted through the anesthetized skin via an intercostal space to just inside the pleural cavity. The speed of removal of the fluid is easily controlled by opening and closing the hemostat.

DISCUSSION

The method has many advantages. It is relatively simple. It does not necessitate the wearing of gloves or the preparation of an elaborate tray. The difficulties often encountered with a leaky stopcock and the resultant pneumothorax are avoided. The fluid that is aspirated is collected under sterile technique and is easily transported to the laboratory for bacteriologic and cellular studies. Our pathologist has found no destruction of cells caused by

From the Department of Surgery, Jefferson Hospital, Roanoke, Va.
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*Prepared and sold by Baxter Laboratories, Inc.

the collection. In fact, he feels that the fluid arrives in the laboratory in better condition than by the routine method of thoracentesis. If, after aspiration of the fluid, an antibiotic is to be injected into the pleural cavity, it is a simple matter to clamp the tubing near the chest, clean a section of this tubing with an antiseptic and insert penicillin and streptomycin into the pleural cavity by injecting the drug into the tubing.

The 1,000 c.c. vacuum bottle is useful not only because it is inexpensive, but because it serves to remind the house staff that this is the maximum amount that usually should be removed at one time from the pleural cavity.

This same apparatus has been used successfully on a number of occasions for performing a paracentesis abdominis and is the method preferred by a number of our house staff.

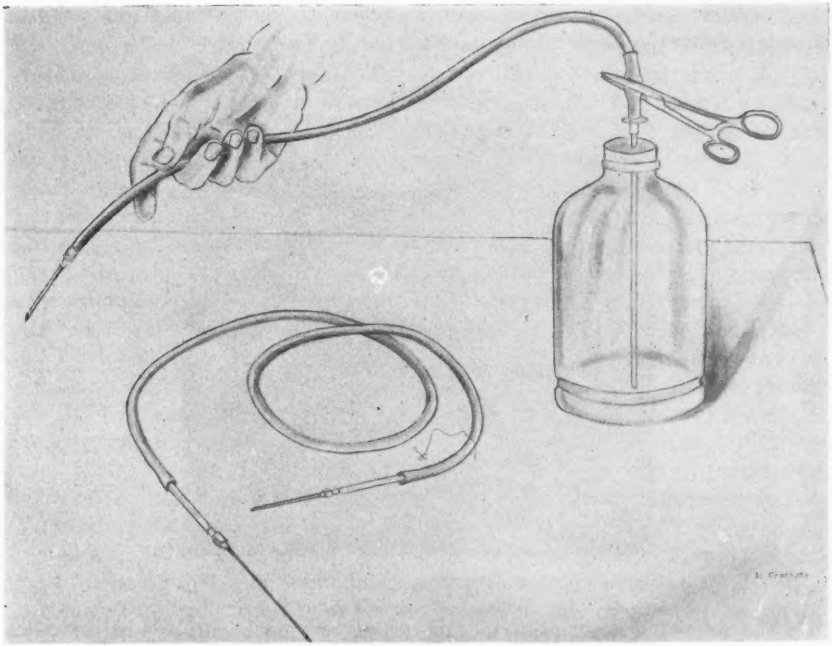

Fig. 1.—The usual donor set tubing is connected to the plasma collecting bottle. The other unit consists of fairly heavy rubber tubing plus two needle adapters and needles of desired length.

This is not as satisfactory to use as the donor tubing.

CONCLUSION

We have re-emphasized a simple method for doing a thoracentesis using a sterile plasma collecting vacuum bottle and a blood donor set. The apparatus is also useful in performing a paracentesis abdominis.

REFERENCE

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A NEONATAL THORACOTOMY RETRACTOR

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THE incidence of congenital atresia of the esophagus has been put at 4 in 10,000* but the increasing recognition of this condition suggests that the figure is in fact much higher.

Most thoracic surgical units of any size deal with a number of these cases each year and the surgical treatment of these now encourages improved technical aids.

A thoracotomy incision in the average newborn infant is seldom more than 2½ by 2 inches and the number of long suture ends disposed around the wound during the anastomosis necessitate a retractor which lies well clear of the operative field, but nevertheless is sufficiently small and strong to give adequate retraction with minimal obstruction to view.

Fig. 1.

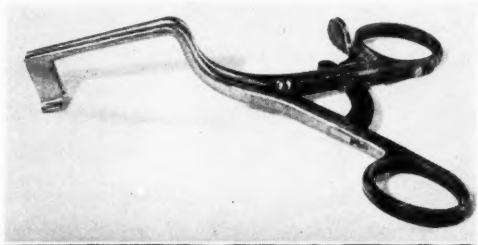

Fig. 2.

The following description of this instrument has been provided by Allen & Hanbury's, Ltd., who made it to my specification and from whom it can be obtained:

> A specially designed stainless steel, cranked, self-retaining rib retractor, fitted with two slightly lipped blades 13/16 inch deep and 5/16 inch wide. Maximum opening of blades 3¾ inches. Total length of retractor 6¾ inches.

I would like to thank Mr. C. Parish, Consultant Thoracic Surgeon, for his advice and encouragement and Miss Westaby of the Medical Photographic Department at Addenbrooke's Hospital for the photographs.

From the Regional Thoracic Surgical Unit, Nelson-Langermann Hospital, Papworth, Cambridge, England.

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*Franklin, R. H.: Congenital Atresia of the Oesophagus, Lancet 2: 243, 1947.

In Memoriam

JOHN ALEXANDER

1891-1954

Notable among the pioneers who have led in the advancement of thoracic surgery was Dr. John Alexander, Professor of Surgery at the University of Michigan, whose death occurred on July 16, 1954. The span of his interests in thoracic surgery, to which his life was steadfastly dedicated, saw it develop from humble beginnings in the early twenties to the full-fledged specialty which it is today. His devotion to thoracic surgery and his remarkable accomplishments in the face of recurring illnesses were an inspiration to all who knew him.

Dr. Alexander was born in Philadelphia on Feb. 24, 1891, the son of Mrs. Mazie Just Alexander and Lucien Hugh Alexander, a prominent lawyer of that city. His preparatory studies at Episcopal and Chestnut Hill Academies were followed by his enrollment in the University of Pennsylvania, where he received the degrees of Bachelor of Science (1912), Master of Arts (1913), and Doctor of Medicine (1916). He was a member of the varsity crew for four years and its captain during his freshman year in the Medical School. Shortly following his graduation he enlisted in an American Unit of the French Army, which had been formed by Dr. Joseph A. Blake. With the entry of the United States into World War I he transferred to the Medical Corps of the United States Army. After the cessation of hostilities he studied at the Université de Lyon where he received a certificate in Surgery and Anatomy in 1919. During this time he had the opportunity of studying with Bérard, who at that time was applying surgical methods for the treatment of pulmonary tuberculosis. Following his return to this country he served for a brief period as Assistant to the Professor of Clinical Surgery at the University of Pennsylvania.

In 1920 Dr. Alexander became a member of the staff of the Surgery Department of the University of Michigan. Several months thereafter he developed a pleural effusion and later a complicating Pott's disease which necessitated a protracted period of rest in Saranac Lake, New York. Perhaps no more stimulating example of devotion to medical science and a will to overcome adversity can be shown than in his writing of the first English text on the surgical treatment of pulmonary tuberculosis. The need for a plaster body cast and the support of a Bradford frame were not deterrents. Not to be overcome by these physical handicaps, he developed a specially designed

support for books which could hold them above his head for greater convenience. After two years there emerged the *Surgery of Pulmonary Tuberculosis,* for which he received the 1925 quinquennial Samuel D. Gross Prize of the Philadelphia Academy of Surgery. This volume was dedicated to the belief that thousands of lives would be lost if surgery were not undertaken for patients with pulmonary tuberculosis. His prophesies were fulfilled by the rapid development of an era of surgical therapy which became an example to the world at large.

John Alexander
(1891-1954)

On recovery from his illness Dr. Alexander returned to the University of Michigan in 1926 and continued with general and thoracic surgery until, in 1928, the number of thoracic surgery patients had increased sufficiently to warrant his full-time preoccupation with this specialty. His first resident in thoracic surgery was appointed in the same year. In 1932 the residency program was augmented to a two-year plan, and his trainees, numbering 79 at the time of his death, were a continuing source of pride and satisfaction.

So rapid were the changes in the surgical treatment of pulmonary tuberculosis in the early thirties and so great became the need for a completely new authoritative text, that Dr. Alexander again presented the subject as the comprehensive surgical classic, *The Collapse Therapy of Pulmonary Tuberculosis,* which appeared in 1937. Although he is particularly well known

for his writings on the subject of pulmonary tuberculosis, Dr. Alexander was instrumental in popularizing the two-stage technique for lobectomy for bronchiectasis at a time when the mortality of lobectomy was almost prohibitive. Using this technique he was able to report the first sizable series of lobectomy cases in America with the then low mortality of 16 per cent. More recently he demonstrated the effectiveness of resection of pulmonary metastases. In the field of cardiovascular diseases his observations on localized stimulation of the exposed human heart led to a reversal in terminology of right and left bundle branch block. He was the first surgeon to resect an aneurysm of the thoracic aorta successfully.

Dr. Alexander was a member of the American Association for Thoracic Surgery and was elected its president in 1935. He was also a member of the American Surgical Association, the Central Surgical Association, the International Society of Surgery, the Society for Clinical Surgery, the American Medical Association, the Michigan State Medical Society, the National Tuberculosis Association, of which he has been a director and vice president, and the American Trudeau Society, of which he was president-elect in 1947, an honor which he had to relinquish because of a recurrence of illness. He was a founder member of the American Board of Surgery and the Board of Thoracic Surgery, a member of the Editorial Boards of the JOURNAL OF THORACIC SURGERY and the *International Abstracts of Surgery*, an honorary member of the Sociedad de Argentina de Cirurjanos, Société Belge de Chirurgie, Société Française de la Tuberculose, Sociedad Paraguaya de Tisiologia, and the Sociedad Ecutoriana de Tisiologia. He was a member of Delta Tau Delta, Nu Sigma Nu, Alpha Omega Alpha, Sigma Xi, and Phi Kappa Phi. Among the awards which Dr. Alexander received were the honorary degree of Doctor of Science conferred upon him by the University of Pennsylvania, the Trudeau Medal of the National Tuberculosis Association, the Bruce H. Douglas Award of the Michigan Trudeau Society, and the Henry Russel Award and the Henry Russel Lectureship of the University of Michigan.

Dr. Alexander was a brilliant scholar and inspiring teacher. His courage, cheerfulness, and optimism in the face of recurring illnesses were a source of wonderment and admiration to his many friends. In his later years his love for the outdoors was evident in his hobbies of gardening and duck hunting which he shared with his wife, the former Emma Ward Woolfolk, to whom he was married in 1936. Although thoracic surgery has lost one of its greatest pioneers, John Alexander will long be remembered by his many friends and students and his noteworthy contributions to thoracic surgery will remain a lasting tribute to his memory.

Cameron Haight.

Announcements

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The Thirty-fifth Annual Meeting of the American Association for Thoracic Surgery will be held April 24, 25, and 26, 1955, in Atlantic City, New Jersey. Headquarters will be at the Chalfonte-Haddon Hall.

Requests for Hotel Reservations

These should be mailed directly to Miss Doris Sears, Convention Manager, Chalfonte-Haddon Hall, Atlantic City, New Jersey. Please mention this Association, the type of accommodation desired, date and approximate hour of arrival. If accommodations are desired elsewhere in Atlantic City, please communicate directly with the hotel of your choice.

Thoracic Surgical Forum

By vote of the membership, the Forum is to be continued for the presentation of short papers on current thoracic research, experimental problems, anatomical studies and modified or new surgical techniques. The inclusion of any considerable amount of clinical material in these reports makes the choice more difficult and the Program Committee reserves the right to reject such papers or to suggest their transferral to the regular program. Papers at this session will be limited sharply to ten minutes, including the showing of lantern slides or other illustrative material. At the discretion of the President, three minutes may be allowed as the maximum discussion for each paper.

These papers will be published together in one issue in the JOURNAL OF THORACIC SURGERY. Maximum length: 3,000 words without illustrations. If illustrated, an appropriate number of words must be subtracted for each cut used.

Abstracts for Papers

Abstracts for the presentation of papers at the 1955 meeting, including papers before the Forum, must be received on or before Dec. 15, 1954; otherwise they will not be considered by the Program Committee.

Abstracts should be labeled "For Thoracic Surgical Forum," or "For Regular Program."

Five, repeat, five copies of each abstract must be sent to the Secretary of the Association, Dr. Paul C. Samson, 3959 Happy Valley Road, Lafayette, California. They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words and accurately reflect the contents of the completed paper.

Applications for Membership

Applications for Associate Membership in the Association must be received by the Membership Committee not later than Dec. 15, 1954; otherwise, the applications will be deferred for consideration until 1956.

Applicants must be sponsored by three Active or Senior members of the Association. In addition to the signatures on the application form, the sponsors must forward a *separate* letter concerning the applicant directly to Dr. Lyman A. Brewer, III, Chairman of the Membership Committee, 2010 Wilshire Blvd., Los Angeles 5, Calif.

Completed application forms and the sponsoring letters *independently* sent by the three sponsors must be received by Dr. Brewer not later than Dec. 15, 1954, to be considered for action in Atlantic City.

Sponsors are reminded that *new* letters of recommendation must be forwarded to the Membership Committee should their applicants be held over for reconsideration a second year.